

in Derwent Patent Files

NEWS 6 Oct 27 Plasdoc Key Serials Dictionary and Echoing added to
Derwent Subscriber Files WPIDS and WPIX

NEWS 7 Nov 29 Derwent announces further increase in updates for DWPI

NEWS 8 Dec 5 French Multi-Disciplinary Database PASCAL Now on STN

NEWS 9 Dec 5 Trademarks on STN - New DEMAS and EUMAS Files

NEWS 10 Dec 15 2001 STN Pricing

NEWS 11 Dec 17 Merged CEABA-VTB for chemical engineering and
biotechnology

NEWS 12 Dec 17 Corrosion Abstracts on STN

NEWS 13 Dec 17 SYNTHLINE from Prous Science now available on STN

NEWS 14 Dec 17 The CA Lexicon available in the CAPLUS and CA files

NEWS 15 Jan 05 MIDSLINE is being removed from STN

NEWS 16 Feb 06 Engineering Information Encompass files have new names

NEWS 17 Feb 16 TOXLINE no longer being updated

NEWS EXPRESS FREE UPGRADE 5.0e FOR STN EXPRESS 5.0 WITH DISCOVER!
(WINDOWS) NOW AVAILABLE

NEWS HOURS STN Operating Hours Plus Help Desk Availability

NEWS INTER General Internet Information

NEWS LOGIN Welcome Banner and News Items

NEWS PHONE Direct Dial and Telecommunication Network Access to STN

NEWS WWW CAS World Wide Web Site (general information)

Enter NEWS followed by the item number or name to see news on that
specific topic.

All use of STN is subject to the provisions of the STN Customer
agreement. Please note that this agreement limits use to scientific
research. Use for software development or design or implementation
of commercial gateways or other similar uses is prohibited and may
result in loss of user privileges and other penalties.

* * * * * STN Columbus * * * * *

FILE 'HOME' ENTERED AT 13:04:40 ON 02 APR 2001

=> file reg

COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	0.15	0.15

FILE 'REGISTRY' ENTERED AT 13:04:49 ON 02 APR 2001

USE IS SUBJECT TO THE TERMS OF YOUR STN CUSTOMER AGREEMENT.

PLEASE SEE "HELP PAGETERMS" FOR DETAILS.

COPYRIGHT (C) 200 American Chemical Society (ACS)

STRUCTURE FILE UPDATES: 1 APR 2001 HIGHEST RN 329683-87-6

DICTIONARY FILE UPDATES: 1 APR 2001 HIGHEST RN 329683-87-6

TSCA INFORMATION NOW CURRENT THROUGH July 8, 2000

Please note that search-term pricing does apply when
conducting Smart SELECT searches.

Structure search limits have been increased. See HELP SLIMIT
for details.

=> s pagoclone/cn

L1 1 PAGACLONE/CN

=> file bioscienc

FILE 'DRUGMONOG' ACCESS NOT AUTHORIZED
COST IN U.S. DOLLARS

SINCE FILE	TOTAL
ENTRY	SESSION
4.11	4.26

FULL ESTIMATED COST

FILE 'ADISALERTS' ENTERED AT 13:05:10 ON 02 APR 2001
COPYRIGHT (C) 2001 Adis International Ltd. (ADIS)

FILE 'ADISINSIGHT' ENTERED AT 13:05:10 ON 02 APR 2001
COPYRIGHT (C) 2001 Adis International Ltd. (ADIS)

FILE 'AGRICOLA' ENTERED AT 13:05:10 ON 02 APR 2001

FILE 'ANABSTR' ENTERED AT 13:05:10 ON 02 APR 2001
COPYRIGHT (c) 2001 THE ROYAL SOCIETY OF CHEMISTRY (RSC)

FILE 'AQUASCI' ENTERED AT 13:05:10 ON 02 APR 2001
(c) 2001 FAO (on behalf of the ASFA Advisory Board) All rights reserved.

FILE 'BIOBUSINESS' ENTERED AT 13:05:10 ON 02 APR 2001
COPYRIGHT (C) 2001 Biological Abstracts, Inc. (BIOSIS)

FILE 'BIOCOMMERCE' ENTERED AT 13:05:10 ON 02 APR 2001
COPYRIGHT (C) 2001 BioCommerce Data Ltd. Richmond Surrey, United Kingdom. All rights reserved

FILE 'BIOSIS' ENTERED AT 13:05:10 ON 02 APR 2001
COPYRIGHT (C) 2001 BIOSIS(R)

FILE 'BIOTECHABS' ACCESS NOT AUTHORIZED

FILE 'BIOTECHDS' ENTERED AT 13:05:10 ON 02 APR 2001
COPYRIGHT (C) 2001 BERWENT INFORMATION LTD

FILE 'BIOTECHNO' ENTERED AT 13:05:10 ON 02 APR 2001
COPYRIGHT (C) 2001 Elsevier Science B.V., Amsterdam. All rights reserved.

FILE 'CABA' ENTERED AT 13:05:10 ON 02 APR 2001
COPYRIGHT (C) 2001 CAB INTERNATIONAL (CABI)

FILE 'CANCERLIT' ENTERED AT 13:05:10 ON 02 APR 2001

FILE 'CAPLUS' ENTERED AT 13:05:10 ON 02 APR 2001
USE IS SUBJECT TO THE TERMS OF YOUR STN CUSTOMER AGREEMENT.
PLEASE SEE "HELP/GETTERMS" FOR DETAILS.
COPYRIGHT (C) 2001 AMERICAN CHEMICAL SOCIETY (ACS)

FILE 'CEABA-VTB' ENTERED AT 13:05:10 ON 02 APR 2001
COPYRIGHT (c) 2001 ECHEMA ev

FILE 'CEN' ENTERED AT 13:05:10 ON 02 APR 2001
COPYRIGHT (C) 2001 American Chemical Society (ACS)

FILE 'CIN' ENTERED AT 13:05:10 ON 02 APR 2001
USE IS SUBJECT TO THE TERMS OF YOUR STN CUSTOMER AGREEMENT.
PLEASE SEE "HELP/GETTERMS" FOR DETAILS.
COPYRIGHT (C) 2001 American Chemical Society (ACS)

FILE 'CONFSCI' ENTERED AT 13:05:10 ON 02 APR 2001
 COPYRIGHT (C) 2001 Cambridge Scientific Abstracts (CSA)

FILE 'CROPB' ENTERED AT 13:05:10 ON 02 APR 2001
 COPYRIGHT (C) 2001 ERWENT INFORMATION LTD

FILE 'CROPU' ENTERED AT 13:05:10 ON 02 APR 2001
 COPYRIGHT (C) 2001 ERWENT INFORMATION LTD

FILE 'DDFB' ACCESS NOT AUTHORIZED

FILE 'DDFU' ACCESS NOT AUTHORIZED

FILE 'DGENE' ENTERED AT 13:05:10 ON 02 APR 2001
 COPYRIGHT (C) 2001 ERWENT INFORMATION LTD

FILE 'DRUGB' ENTERED AT 13:05:10 ON 02 APR 2001
 COPYRIGHT (C) 2001 ERWENT INFORMATION LTD

FILE 'DRUGLAUNCH' ENTERED AT 13:05:10 ON 02 APR 2001
 COPYRIGHT (C) 2001 ASWORLD Publications Ltd

FILE 'DRUGMONOG2' ENTERED AT 13:05:10 ON 02 APR 2001
 COPYRIGHT (C) 2001 ASWORLD Publications Ltd

FILE 'DRUGNL' ENTERED AT 13:05:10 ON 02 APR 2001
 COPYRIGHT (C) 2001 ASWORLD Publications Ltd

FILE 'DRUGU' ENTERED AT 13:05:10 ON 02 APR 2001
 COPYRIGHT (C) 2001 ERWENT INFORMATION LTD

FILE 'DRUGUPDATE' ENTERED AT 13:05:10 ON 02 APR 2001
 COPYRIGHT (C) 2001 ASWORLD Publications Ltd

FILE 'EMBAL' ENTERED AT 13:05:10 ON 02 APR 2001
 COPYRIGHT (C) 2001 Elsevier Science B.V. All rights reserved.

FILE 'EMBASE' ENTERED AT 13:05:10 ON 02 APR 2001
 COPYRIGHT (C) 2001 Elsevier Science B.V. All rights reserved.

FILE 'ESBIOBASE' ENTERED AT 13:05:10 ON 02 APR 2001
 COPYRIGHT (C) 2001 Elsevier Science B.V., Amsterdam. All rights reserved.

FILE 'FOMAD' ENTERED AT 13:05:10 ON 02 APR 2001
 COPYRIGHT (C) 2001 Fatherhead Food Research Association

FILE 'FOREGE' ENTERED AT 13:05:10 ON 02 APR 2001
 COPYRIGHT (C) 2001 Fatherhead Food Research Association

FILE 'FROSTI' ENTERED AT 13:05:10 ON 02 APR 2001
 COPYRIGHT (C) 2001 Fatherhead Food Research Association

FILE 'FSTA' ENTERED AT 13:05:10 ON 02 APR 2001
 COPYRIGHT (C) 2001 International Food Information Service

FILE 'GENBANK' ENTERED AT 13:05:10 ON 02 APR 2001

FILE 'HEALSAFE' ENTERED AT 13:05:10 ON 02 APR 2001
 COPYRIGHT (C) 2001 Cambridge Scientific Abstracts (CSA)

FILE 'IFIPAT' ENTERED AT 13:05:10 ON 02 APR 2001
 COPYRIGHT (C) 2001 IFI CLAIMS(R) Patent Services (IFI)

FILE 'JICST-EPLU' ENTERED AT 13:05:10 ON 02 APR 2001
 COPYRIGHT (C) 2001 Japan Science and Technology Corporation (JST)

FILE 'KOSMET' ENTERED AT 13:05:10 ON 02 APR 2001
 COPYRIGHT (C) 2001 International Federation of the Societies of Cosmetics Chemists

FILE 'LIFESCI' ENTERED AT 13:05:10 ON 02 APR 2001
 COPYRIGHT (C) 2001 Cambridge Scientific Abstracts (CSA)

FILE 'MEDICONF' ENTERED AT 13:05:10 ON 02 APR 2001
 COPYRIGHT (c) 2001 AIRBASE Datenbank GmbH, Hannover, Germany

FILE 'MEDLINE' ENTERED AT 13:05:10 ON 02 APR 2001

FILE 'NIOSHTIC' ENTERED AT 13:05:10 ON 02 APR 2001
 COPYRIGHT (C) 2001 U.S. Secretary of Commerce on Behalf of the U.S. Government

FILE 'NTIS' ENTERED AT 13:05:10 ON 02 APR 2001
 Compiled and distributed by the NTIS, U.S. Department of Commerce.
 It contains copyrighted material.
 All rights reserved. (2001)

FILE 'OCEAN' ENTERED AT 13:05:10 ON 02 APR 2001
 COPYRIGHT (C) 2001 Cambridge Scientific Abstracts (CSA)

FILE 'PASCAL' ENTERED AT 13:05:10 ON 02 APR 2001
 Any reproduction, dissemination in part or in full,
 by means of any process and on any support whatsoever
 is prohibited without the prior written agreement of INIST-CNRS.
 COPYRIGHT (C) 2001 INIST-CNRS. All rights reserved.

FILE 'PHAR' ENTERED AT 13:05:10 ON 02 APR 2001
 COPYRIGHT (C) 2001 PJB Publications Ltd. (PJB)

FILE 'PHIC' ENTERED AT 13:05:10 ON 02 APR 2001
 COPYRIGHT (C) 2001 PJB Publications Ltd. (PJB)

FILE 'PHIN' ENTERED AT 13:05:10 ON 02 APR 2001
 COPYRIGHT (C) 2001 PJB Publications Ltd. (PJB)

FILE 'PROMT' ENTERED AT 13:05:10 ON 02 APR 2001
 COPYRIGHT (C) 2001 PJB Publications Ltd. (PJB)

FILE 'PROMT' ENTERED AT 13:05:10 ON 02 APR 2001
 COPYRIGHT (C) 2001 PJB Publications Ltd. (PJB)

FILE 'SCISEARCH' ENTERED AT 13:05:10 ON 02 APR 2001
 COPYRIGHT (C) 2001 Institute for Scientific Information (ISI) (R)

FILE 'SYNTHLINE' ENTERED AT 13:05:10 ON 02 APR 2001
 COPYRIGHT (C) 2001 PJB Publications Ltd. (PJB)

FILE 'TOXLINE' ENTERED AT 13:05:10 ON 02 APR 2001

FILE 'TOXLIT' ENTERED AT 13:05:10 ON 02 APR 2001

FILE 'USPATFULL' INDEXED AT 13:05:10 ON 02 APR 2001
CA INDEXING COPY (C) 2001 AMERICAN CHEMICAL SOCIETY (ACS)

FILE 'WPIDS' ENDED AT 13:05:10 ON 02 APR 2001
COPYRIGHT (C) 2001 ERWENT INFORMATION LTD

FILE 'WPINDEX' NOT AUTHORIZED

=> s l1 or pagoclon or (rp 59037) or (rp 62955)

'CN' IS NOT A VALID FIELD CODE

'CN' IS NOT A VALID FIELD CODE

13 FILES SEARCHED

'CN' IS NOT A VALID FIELD CODE

'CN' IS NOT A VALID FIELD CODE

'CN' IS NOT A VALID FIELD CODE

'CN' IS NOT A VALID FIELD CODE

'CN' IS NOT A VALID FIELD CODE

'CN' IS NOT A VALID FIELD CODE

'CN' IS NOT A VALID FIELD CODE

27 FILES SEARCHED

'CN' IS NOT A VALID FIELD CODE

'CN' IS NOT A VALID FIELD CODE

'CN' IS NOT A VALID FIELD CODE

'CN' IS NOT A VALID FIELD CODE

'CN' IS NOT A VALID FIELD CODE

'CN' IS NOT A VALID FIELD CODE

'CN' IS NOT A VALID FIELD CODE

'CN' IS NOT A VALID FIELD CODE

'CN' IS NOT A VALID FIELD CODE

'CN' IS NOT A VALID FIELD CODE

'CN' IS NOT A VALID FIELD CODE

45 FILES SEARCHED

'CN' IS NOT A VALID FIELD CODE

L2 152 PAGOCLONE OR (RP 59037) OR (RP 62955)

=> duplicate

ENTER REMOVE, COPY, ONLY, OR (?):remove

ENTER L# LIST (0):12

DUPLICATE IS NOT ALLOWED IN 'ADISINSIGHT, BIOCOMMERCE, DGENE, DRUGLAUNCH, DRUGMONOG2, DRUGS, FOREGE, GENBANK, KOSMET, MEDICONF, PHAR, SYNTHLINE'. ANSWERS FROM THESE FILES WILL BE CONSIDERED UNIQUE

DUPLICATE PREFERENCE IS 'ADISALERTS, ADISINSIGHT, BIOCOMMERCE, BIOSIS, BIOTECHNO, CAPL, CABA-VTB, CIN, DRUGNL, DRUGU, DRUGUPDATES, EMBASE, MEDLINE, PASCAL, PHIN, PROMT, SCISEARCH, TOXLINE, TOXLIT, USPATFULL' MORE THAN ONE FILE? Y/(N):n

PROCESSING COMPLETE FOR L2

L3 130 DUPLICATE REMOVE L2 (22 DUPLICATES REMOVED)

=> s stutter? or ch dysfluen? or dysarthr? or speech block? or logospasm or tourette

17 FILES SEARCHED

30 FILES SEARCHED

45 FILES SEARCHED

L4 32929 STUTTER? OR SPEECH DYSFLUEN? OR DYSARTH? OR SPEECH BLOCK? OR LOGOASM OR TOURETTE

=> s l3 and l4

29 FILES SEARCHED

L5 2 L4

=> d ibib abs

L5 ANSWER 1 CAPLUS COPYRIGHT 2001 ACS
ACCESSION NUMBER: 2001:100965 CAPLUS
DOCUMENT NUMBER: 134:141757
TITLE: Methods and compositions using GABA receptor
modulators for alleviating **stuttering**
INVENTOR(S): Murphy, John J.; D'orlando, Kay Jorgenson
PATENT ASSIGNEE: Interneuron Pharmaceuticals, Inc., USA
SOURCE: PCT Int. Appl., 22 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUMBER: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2001008	A2	20010208	WO 2000-US20402	20000727
W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR, CU, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, UZ, VN, YU, ZA, ZW, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GR, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
PRIORITY APPLN.:			US 1999-362691	19990729
OTHER SOURCE(S):		MARPAT 134:141757		
AB	Methods of	ing stuttering include treating people with		
	.gamma.-a	amic acid (GABA) receptor modulators, including		
	cyclopyrrolone	A second active agent may be used with GABA receptor		
	modulators	ive enantiomers, active metabolites, and pharmaceutically		
	acceptable	of GABA receptor modulators, including cyclopyrrolones,		
	are accept	ponents of the compns. The cyclopyrrolone class of		
	modulators	es pagoclone , suriclone, zopiclone,		
	2-(7-chloro	hthyridin-1,8-yl)-3-(5-methyl-2-oxohexyl)isoindolin-1-		
	one, 2-(7-	-2-naphthyridin-1,8-yl)isoindolin-1-yl-4-		
	acetamido	, and 2-(7-chloro-1,8-naphthyridin-2yl)-3-(5-methyl-5-		
	hydroxy-2-	yl)-1-isoindolinone.		
TI	Methods a	ositions using GABA receptor modulators for alleviating		
	stuttering			
AB	Methods of	ing stuttering include treating people with		
	.gamma.-a	amic acid (GABA) receptor modulators, including		
	cyclopyrrolone	A second active agent may be used with GABA receptor		
	modulators	ive enantiomers, active metabolites, and pharmaceutically		
	acceptable	of GABA receptor modulators, including cyclopyrrolones,		
	are accept	ponents of the compns. The cyclopyrrolone class of		
	modulators	es pagoclone , suriclone, zopiclone,		
	2-(7-chloro	hthyridin-1,8-yl)-3-(5-methyl-2-oxohexyl)isoindolin-1-		
	one, 2-(7-	-2-naphthyridin-1,8-yl)isoindolin-1-yl-4-		
	acetamido	, and 2-(7-chloro-1,8-naphthyridin-2yl)-3-(5-methyl-5-		
	hydroxy-2-	yl)-1-isoindolinone.		
ST	GABA rece	ulator stuttering treatment; cyclopyrrolone		
	compd stu	treatment		
IT	Drug deli	tems		
	(GABA	modulators for alleviating stuttering)		
IT	GABA rece			
RL: BPR		l process); BSU (Biological study, unclassified); BIOL		

	(Biologic	; PROC (Process)
	(GABA	modulators for alleviating stuttering)
IT	GABA agon	
	(GABA	receptor modulators for alleviating stuttering)
IT	GABA recep	
	RL: BPR	al process); BSU (Biological study, unclassified); BIOL
	(Biologic	; PROC (Process)
	(GABA	receptor modulators for alleviating stuttering)
IT	Brain, d	
	(Gille	Tourette syndrome; GABA receptor modulators for
	allevi	stuttering)
IT	Drug deliv	tems
	(buccal	receptor modulators for alleviating stuttering)
IT	Proteins,	c or class
	RL: BAC	al activity or effector, except adverse); THU
	(Therape	; BIOL (Biological study); USES (Uses)
	(diazep	ing inhibitory protein and fragments; GABA receptor
	modulat	alleviating stuttering)
IT	Nervous s	
	(disea	tering; GABA receptor modulators for
	allevi	stuttering)
IT	Drug deliv	tems
	(epidur	A receptor modulators for alleviating stuttering
)	
IT	Drug deliv	tems
	(inject	m.; GABA receptor modulators for alleviating
	stutter	
IT	Drug deliv	tems
	(inject	v.; GABA receptor modulators for alleviating
	stutter	
IT	Drug deliv	tems
	(inject	c.; GABA receptor modulators for alleviating
	stutter	
IT	Drug deliv	tems
	(intrac	entricular; GABA receptor modulators for alleviating
	stutter	
IT	Drug deliv	tems
	(intrac	GABA receptor modulators for alleviating
	stutter	
IT	Behavior	
	(motor	er, motor tic; GABA receptor modulators for alleviating
	stutter	
IT	Drug deliv	tems
	(nasal;	receptor modulators for alleviating stuttering)
IT	Drug deliv	tems
	(oral;	receptor modulators for alleviating stuttering)
IT	Drug deliv	tems
	(parent	GABA receptor modulators for alleviating
	stutter	
IT	Drug deliv	tems
	(recta	receptor modulators for alleviating stuttering)
IT	Disease,	
	(speed	; GABA receptor modulators for alleviating
	stutter	
IT	Drug deliv	tems
	(transd	GABA receptor modulators for alleviating
	stutter	
IT	Drug deliv	tems
	(vagin	receptor modulators for alleviating stuttering
)	

IT 50-06-6, Phenobarbital, biological studies 50-06-6D, Phenobarbital, enantiomers and metabolites 53-43-0, Dehydroepiandrosterone, enantiomers and metabolites 57-43-2, Amobarbital 57-43-2D, Amobarbital, enantiomers and metabolites 57-83-0, Progesterone, biological studies 57-83-0D, Progesterone, enantiomers and metabolites 58-25-3, Chlordiazepoxide 58-25-3D, Chlordiazepoxide, enantiomers and metabolites 76-73-3, Secobarbital 76-73-3D, Secobarbital, enantiomers and metabolites 76-74-4, Pentobarbital 76-74-4D, Pentobarbital, enantiomers and metabolites 76-75-5, Thiopental 76-75-5D, Thiopental, enantiomers and metabolites 77-02-1, Aprobital 77-02-1D, Aprobital, enantiomers and metabolites 77-26-9, Butabital 77-26-9D, Butabital, enantiomers and metabolites 115-38-8, Mephobarbital 115-38-8D, Mephobarbital, enantiomers and metabolites 125-40-6, Butabarbital 125-40-6D, Butabarbital, enantiomers and metabolites 145-13-1, Pregnenolone 145-13-1D, Pregnenolone, enantiomers and metabolites 151-83-7, Methohexital 151-83-7D, Methohexital, enantiomers and metabolites 439-14-5, Diazepam 439-14-5D, Diazepam, enantiomers and metabolites 485-49-4, Bicuculline 485-49-4D, Bicuculline, enantiomers and metabolites 516-54-1, Allopregnanolone 516-54-1D, Allopregnanolone, enantiomers and metabolites 516-55-2, Allopregnanolone 516-55-2D, Allopregnanolone, enantiomers and metabolites 567-03-3, Tetrahydrodeoxycorticosterone 567-03-3D, Tetrahydrodeoxycorticosterone, enantiomers and metabolites 604-75-1, Oxazepam 604-75-1D, Oxazepam, enantiomers and metabolites 846-49-1, Lorazepam 846-49-1D, Lorazepam, enantiomers and metabolites 846-50-4, Temazepam 846-50-4D, Temazepam, enantiomers and metabolites 1005-93-2, Etbicuphat 1005-93-2D, Etbicuphat, enantiomers and metabolites 1134-47-0, Baclofen 1134-47-0D, Baclofen, enantiomers and metabolites 1449-89-4, Mebicuphat 1449-89-4D, Mebicuphat, enantiomers and metabolites 1622-62-4, Flunitrazepam 1622-62-4D, Flunitrazepam, enantiomers and metabolites 2078-54-8, Propofol 2078-54-8D, Propofol, enantiomers and metabolites 2955-38-6, Prazepam 2955-38-6D, Prazepam, enantiomers and metabolites 3289-22-3, Flucyben 3289-22-3D, Flucyben, enantiomers and metabolites 4406-37-5, Pregnanolone 4406-37-5D, Pregnanolone, enantiomers and metabolites 17617-23-1, Flurazepam 17617-23-1D, Flurazepam, enantiomers and metabolites 17617-45-7, Picrotoxinin 17617-45-7D, Picrotoxinin, enantiomers and metabolites 21416-53-5, Picrotin 21416-53-5D, Picrotin, enantiomers and metabolites 23092-17-3, Halazepam 23092-17-3D, Halazepam, enantiomers and metabolites 23930-19-0, Triazolam 23930-19-0D, Triazolam, enantiomers and metabolites 28911-01-5, Alprazolam 28911-01-5D, Alprazolam, enantiomers and metabolites 29617-43-4, Estazolam 29617-43-4D, Estazolam, enantiomers and metabolites 33125-97-2, Etomidate 33125-97-2D, Etomidate, enantiomers and metabolites 34985-87-0, Chlorazepam 34985-87-0D, Chlorazepam, enantiomers and metabolites 36104-80-0, Camazepam 36104-80-0D, Camazepam, enantiomers and metabolites 36735-22-5, Quazepam 36735-22-5D, Quazepam, enantiomers and metabolites 43200-80-2, Zopiclone 43200-80-2D, Zopiclone, enantiomers and metabolites 51052-72-3, Isobicyclazepam 51052-72-3D, Isobicyclazepam, enantiomers and metabolites 51486-74-9, Propylbicyclazepam 51486-74-9D, Propylbicyclazepam, enantiomers and metabolites 52463-83-9, Pinazepam 52463-83-9D, Pinazepam, enantiomers and metabolites 53813-83-5, Suriclone 53813-83-5D, Suriclone, enantiomers and metabolites 57109-90-7, Chlorazepate 57109-90-7D, Chlorazepate, enantiomers and metabolites 59467-70-8, Midazolam 59467-70-8D, Midazolam, enantiomers and metabolites 109370-34-5, Avermectin B

109370-34-5, Suriclon B, enantiomers and metabolites 117705-18-7
 117705-18-7, Suriclon B, enantiomers and metabolites 133737-32-3,
Pagoclone 133737-48-1, **Pagoclone**, enantiomers
 and metabolites 133737-48-1 133737-48-1D, enantiomers and metabolites
 153046-19-6, Suriclon B, enantiomers and metabolites 224790-70-9,
 Cloflubicyne, 224790-70-9D, Cloflubicyne, enantiomers and metabolites
 224790-71-0, Etbicthythionat 224790-71-0D, Etbicthythionat, enantiomers and
 metabolites
 RL: BAC (Biological activity or effector, except adverse); THU
 (Therapeutic use); BIOL (Biological study); USES (Uses)
 (GABA receptor modulators for alleviating **stuttering**)

L5 ANSWER 2 OF 3 LIT
 ACCESSION NUMBER: 1:3118 TOXLIT
 DOCUMENT NUMBER: 134-141757Z
 TITLE: Methods and compositions using GABA receptor modulators
 for

alleviating **stuttering**.
 AUTHOR: Murphy JJ; D'orlando KJ
 SOURCE: (1). PCT Int. Appl. PATENT NO. 018670 02/08/2001
 (Interneuron Pharmaceuticals, Inc.).
 EN: PIXXD2.

PUB. COUNTRY: UNITED STATES
 DOCUMENT TYPE: Patent
 FILE SEGMENT:
 LANGUAGE: English
 OTHER SOURCE: 134:141757
 ENTRY MONTH: 03

AB Methods of treating **stuttering** include treating people with
 .gamma.-aminobutyric acid (GABA) receptor modulators, including
 cyclopyrrolones. A second active agent may be used with GABA receptor
 modulators. Enantiomers, active metabolites, and pharmaceutically
 acceptable salts of GABA receptor modulators, including cyclopyrrolones,
 are acceptable components of the compns. The cyclopyrrolone class of
 modulators includes **pagoclone**, suriclone, zopiclone,
 2-(7-chloro-1,8-naphthyridin-1,8-yl)-3-(5-methyl-2-oxohexyl)isoindolin-1-
 one, 2-(7-chloro-1,8-naphthyridin-1,8-yl)isoindolin-1-yl-4-
 acetamidobutyl, and 2-(7-chloro-1,8-naphthyridin-2yl)-3-(5-methyl-5-
 hydroxy-1-oxohexyl)-1-isoindolinone.

TI Methods and compositions using GABA receptor modulators for alleviating
stuttering.

AB Methods of treating **stuttering** include treating people with
 .gamma.-aminobutyric acid (GABA) receptor modulators, including
 cyclopyrrolones. A second active agent may be used with GABA. . .
 acceptable salts of GABA receptor modulators, including cyclopyrrolones,
 are acceptable components of the compns. The cyclopyrrolone class of
 modulators includes **pagoclone**, suriclone, zopiclone,
 2-(7-chloro-1,8-naphthyridin-1,8-yl)-3-(5-methyl-2-oxohexyl)isoindolin-1-
 one, 2-(7-chloro-1,8-naphthyridin-1,8-yl)isoindolin-1-yl-4-
 acetamidobutyl, and 2-(7-chloro-1,8-naphthyridin-2yl)-3-(5-methyl-5-
 hydroxy-1-oxohexyl)-1-isoindolinone.

ST GABA receptor modulator **stuttering** treatment; cyclopyrrolone
 compd stuttt treatment

=> s gaba or gamma-aminobutyric acid

11 FILES SEARCHED
 20 FILES SERIALIZED
 29 FILES SELECTED
 39 FILES SEARCHED

52 FILES SEARCHED
L6 2408 L6 GAMMA AMINO BUTYRIC ACID

=> s 16 and 17
45 FILES SEARCHED
L7 147 L7

=> duplicate
ENTER REMOVE, ID ONLY, OR (?):remove
ENTER L# LIST OF 17
DUPLICATE IS 17 FILE IN 'ADISINSIGHT, BIOCOMMERCE, DGENE, DRUGLAUNCH,
DRUGMONOG2, DRUG, FOREGE, GENBANK, KOSMET, MEDICONF, PHAR, SYNTHLINE'.
ANSWERS FROM THE 17 WILL BE CONSIDERED UNIQUE
DUPLICATE PREFER 'ADISALERTS, ADISINSIGHT, BIOSIS, CAPLUS, DRUGB,
DRUGU, EMBAL, EMBASE, BIOBASE, JICST-EPLUS, MEDLINE, PASCAL, PHIN, PROMT,
SCISEARCH, TOXLIT, USPATFULL, WPIDS'
KEEP DUPLICATES MORE THAN ONE FILE? Y/(N):n
PROCESSING COMPLETE FOR L7
L8 147 L8 DATE REMOVE L7 (27 DUPLICATES REMOVED)

=> s 16 (s) 17
38 FILES SEARCHED
L9 147 L9

=> duplicate
ENTER REMOVE, ID ONLY, OR (?):remove
ENTER L# LIST OF 19
DUPLICATE IS 19 FILE IN 'ADISINSIGHT, BIOCOMMERCE, DGENE, DRUGLAUNCH,
DRUGMONOG2, DRUG, FOREGE, GENBANK, KOSMET, MEDICONF, PHAR, SYNTHLINE'.
ANSWERS FROM THE 19 WILL BE CONSIDERED UNIQUE
DUPLICATE PREFER 'BIOSIS, CAPLUS, DRUGB, DRUGU, EMBAL, EMBASE,
JICST-EPLUS, MEDLINE, PASCAL, SCISEARCH, TOXLINE, TOXLIT, USPATFULL, WPIDS'
KEEP DUPLICATES MORE THAN ONE FILE? Y/(N):n
PROCESSING COMPLETE FOR L9
L10 147 L10 DATE REMOVE L9 (16 DUPLICATES REMOVED)

=> d ti 1-5

L10 ANSWER 1 OF 1 PLUS COPYRIGHT 2001 ACS
TI Methods and conditions using GABA receptor modulators for
alleviating anxiety

L10 ANSWER 1 OF 1 TOXLIT
TI Methods and conditions using GABA receptor modulators for
alleviating anxiety.

L10 ANSWER 1 OF 1 USPATFULL
TI Guanidine heterocycle compounds useful as alpha-2 adrenoceptor
agonists

L10 ANSWER 1 OF 1 EMBAL COPYRIGHT 2001 ELSEVIER SCI. B.V. DUPLICATE 1
TI Baclofen in Tourette syndrome: A double-blind,
placebo-controlled, crossover trial.

L10 ANSWER 1 OF 1 PLUS COPYRIGHT 2001 ACS DUPLICATE 2
TI Treatment of post-traumatic stress disorder, obsessive-compulsive
disorder
and related psychiatric disorders

=> d ti 5-45

- L10 ANSWER 05 PLUS COPYRIGHT 2001 ACS DUPLICATE 2
TI Treatment of traumatic stress disorder, obsessive-compulsive disorder and related psychiatric disorders
- L10 ANSWER 01 PATFULL
TI 2-imino-1-methyl-5H-indole compounds useful as alpha-2 adrenoceptor agonists
- L10 ANSWER 01 PATFULL
TI Allele-specific diagnosis of reward deficiency syndrome and treatment
- L10 ANSWER 05 PATFULL
TI 6-(2-amino-1-methyl-5H-indol-3-yl)quinoxaline compounds useful as alpha-2 adrenoceptor agonists
- L10 ANSWER 01 PATFULL
TI 2-imino-1-methyl-5H-benzoxazole compounds useful as alpha-2 adrenoceptor agonists
- L10 ANSWER 01 PATFULL
TI Methods for treating tardive dyskinesia and other movement disorders using GABA-A receptor antagonists
- L10 ANSWER 01 IDS COPYRIGHT 2001 DERWENT INFORMATION LTD
TI Identification of interest and relevance to neurological disorders or dysfunction as Parkinson's disease involves use of biological array including those associated with neurotransmitter molecules.
- L10 ANSWER 01 IDS COPYRIGHT 2001 DERWENT INFORMATION LTD
TI Treating movement disorders using agents which increase GABA-A neurotransmission and decrease NMDA-glutamate neurotransmission.
- L10 ANSWER 01 BASE COPYRIGHT 2001 ELSEVIER SCI. B.V.
TI Reward deficiency syndrome: A biogenetic model for the diagnosis and treatment of obsessive, addictive, and compulsive behaviors.
- L10 ANSWER 01 BIOSIS COPYRIGHT 2001 BIOSIS
TI Reward deficiency syndrome: A biogenetic model for the diagnosis and treatment of obsessive, addictive, and compulsive behaviors.
- L10 ANSWER 01 PLUS COPYRIGHT 2001 ACS DUPLICATE 3
TI Methods for treating movement disorders simultaneously acting as NMDA-type glutamate receptor antagonists and GABA-A receptor agonists for treating tardive dyskinesia and other movement disorders
- L10 ANSWER 01 OXLIT
TI Methods for treating movement disorders simultaneously acting as NMDA-type glutamate receptor antagonists and GABA-A receptor agonists for treating tardive dyskinesia and other movement disorders.
- L10 ANSWER 01 PATFULL
TI 2-Imino-1-methyl-5H-indole heterocyclic compounds useful as alpha-2 adrenoceptor agonists
- L10 ANSWER 01 PATFULL

TI 7-(2-amino)quinoline compounds useful as alpha-2
adren

L10 ANSWER 1 USPATFULL
TI 2-imino heterocyclic compounds useful as alpha-2
adren

L10 ANSWER 1 USPATFULL
TI 6-(2-amino) quinolines useful as alpha-2 adrenoceptor
agonis

L10 ANSWER 2 USPATFULL
TI Use of in treating psychiatric disorders

L10 ANSWER 1 BIOSIS COPYRIGHT 2001 DERWENT INFORMATION LTD
TI Treatment of A-uptake related disorders - used for e.g. cluster
headache, alcohol withdrawal symptoms, spasticity, growth
disturbance, chorea dyskinesia or alcohol abuse.

L10 ANSWER 1 BIOSIS COPYRIGHT 2001 BIOSIS DUPLICATE 4
TI Autoantibodies to glutamate decarboxylase in a patient with spinocerebellar
degenera

L10 ANSWER 1 BIOSIS COPYRIGHT 2001 INIST-CNRS. ALL RIGHTS RESERVED.
TIEN Association of a Tourette-like syndrome with ofloxacin

L10 ANSWER 1 BIOSIS COPYRIGHT 2001 DERWENT INFORMATION LTD
TI Combination of sugars and amino acids, opt with other drugs - for
treatment of Alzheimer's disease, depression, hair loss, cancers,
hyperten

L10 ANSWER 2 BIOSIS COPYRIGHT 2001 BIOSIS DUPLICATE 5
TI Neurochemical and some related psychopharmacological aspects of
Tourette's
syndrome

L10 ANSWER 1 MEDLINE DUPLICATE 6
TI Resolution of dysarthria in multiple sclerosis by treatment with weak
electromagnetic fields.

L10 ANSWER 1 BIOSIS COPYRIGHT 2001 DERWENT INFORMATION LTD
TI Pharmacology of Controversy of CNS Stimulants in Gilles de la Tourette's
Syndrome

L10 ANSWER 1 BIOSIS COPYRIGHT 2001 DERWENT INFORMATION LTD
TI Neurotoxicity of beta-Lactam Antibiotics: Predisposing Factors and
Pathogen

L10 ANSWER 1 BIOSIS COPYRIGHT 2001 DERWENT INFORMATION LTD
TI Central Nervous System-Effects of Various Antibacterial Compounds.

L10 ANSWER 1 BIOSIS COPYRIGHT 2001 DERWENT INFORMATION LTD
TI The Effect of Different Vigabatrin Treatment Regimens on CSF
Biochemistry
and Seizure Control in Epileptic Patients.

L10 ANSWER 1 BIOSIS COPYRIGHT 2001 DERWENT INFORMATION LTD
TI A Case of Lactic Acidemia Type I: Effect of Riboflavin and Carnitine.

L10 ANSWER 1 BIOSIS COPYRIGHT 2001 DERWENT INFORMATION LTD

TI Drugs Affecting Movement Disorders.

L10 ANSWER 3 BIOSIS COPYRIGHT 2001 BIOSIS DUPLICATE 7
 TI VIGABATRIN THE TREATMENT OF EPILEPSY A DOUBLE-BLIND PLACEBO-CONTROLLED STUDY.

L10 ANSWER 15 DRUGU COPYRIGHT 2001 DERWENT INFORMATION LTD
 TI Anxiolytic

L10 ANSWER 3 ELSEVIER COPYRIGHT 2001 ELSEVIER SCI. B.V. DUPLICATE 8
 TI Gamma-aminobutyric acid treatment of tardive dyskinesia and other movement disorders.

L10 ANSWER 15 PASCAL COPYRIGHT 2001 INIST-CNRS. ALL RIGHTS RESERVED.
 TIEN Gamma-aminobutyric acid treatment of tardive dyskinesia and other movement disorders.

L10 ANSWER 3 ELSEVIER COPYRIGHT 2001 ELSEVIER SCI. B.V. DUPLICATE 9
 TI Progabide treatment of hyperkinetic extrapyramidal movement disorders.

L10 ANSWER 3 BIOSIS COPYRIGHT 2001 BIOSIS DUPLICATE 10
 TI GLUTARIC ACIDuria TYPE I PRESENTING WITH HYPOGLYCEMIA.

L10 ANSWER 3 BIOSIS COPYRIGHT 2001 BIOSIS
 TI CONCENTRATION OF GAMMA AMINO BUTYRIC-ACID AND ADENOSINE IN THE CEREBROSPINAL FLUID IN PROGRESSIVE MYOCLONUS EPILEPSY WITHOUT LAFORA BODIES.

L10 ANSWER 5 DRUGU COPYRIGHT 2001 DERWENT INFORMATION LTD
 TI Clonazepam Tourette's Syndrome.

L10 ANSWER 4 BIOSIS COPYRIGHT 2001 BIOSIS
 TI PHYSIOLOGICAL CONSIDERATIONS AND A PHARMACOLOGICAL APPROACH IN THE PATHOLOGY OF THE EXTRAPYRAMIDAL SYSTEM.

L10 ANSWER 15 DRUGB COPYRIGHT 2001 DERWENT INFORMATION LTD
 TI CLINICAL MANIFESTATIONS OF THE INITIAL PERIOD OF STUTTERING IN CHILDREN.

L10 ANSWER 5 DRUGB COPYRIGHT 2001 DERWENT INFORMATION LTD
 TI AMINOCLOTRIPINE A NEUROTROPIC AGENT./RUSS./.

L10 ANSWER 5 DRUGU COPYRIGHT 2001 DERWENT INFORMATION LTD
 TI Neurotransmitters and CNS Disease. Basal Ganglia Disease.

=> d ibib a's -45

L10 ANSWER 1 CALS COPYRIGHT 2001 ACS
 ACCESSION NUMBER 201:100965 CAPLUS
 DOCUMENT NUMBER 34:141757
 TITLE: Methods and compositions using GABA receptor modulators for alleviating stuttering
 INVENTOR(S): Murphy, John J.; D'orlando, Kay Jorgenson
 PATENT ASSIGNED: Interneuron Pharmaceuticals, Inc., USA
 SOURCE: CT Int. Appl., 22 pp.
 CODEN: PIXXD2
 DOCUMENT TYPE: Patent
 LANGUAGE: English
 FAMILY ACC. NUMBER: NT:

PATENT INFORMATION

PATENT NO.	INVENTOR	DATE	APPLICATION NO.	DATE
WO 20010208		20010208	WO 2000-US20402	20000727
W:	AL, AR, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, IN, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, UZ, VN, YU, ZA, ZW			
RW:	KE, S, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, ES, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG			
PRIORITY APPL			US 1999-362691	19990729
OTHER SOURCE		ERPAT 134:141757		
AB	Methods for treating stuttering include treating people with .gamma.-aminobutyric acid (GABA) receptor modulators, including cyclopyrrolones. A second active agent may be used with GABA receptor modulators. The active agents, active metabolites, and pharmaceutically acceptable salts of GABA receptor modulators, including cyclopyrrolones, are acceptable components of the compns. The cyclopyrrolone class of modulators includes zopiclone, suriclone, zopiclone, 2-(7-chloro-2-naphthyl)-3-(5-methyl-2-oxohexyl)isoindolin-1-one, 2-(7-chloro-2-naphthyl)-3-(5-methyl-2-oxohexyl)isoindolin-1-yl-4-acetamidobutyrate, and 2-(7-chloro-2-naphthyl)-3-(5-methyl-5-hydroxy-2-oxohexyl)-1-isoindolin-1-yl-4-acetamidobutyrate.			
TI	Methods for alleviating stuttering using GABA receptor modulators for			
AB	Methods for treating stuttering include treating people with .gamma.-aminobutyric acid (GABA) receptor modulators, including cyclopyrrolones. A second active agent may be used with GABA receptor modulators. The active agents, active metabolites, and pharmaceutically acceptable salts of GABA receptor modulators, including cyclopyrrolones, are acceptable components of the compns. The cyclopyrrolone class of modulators includes zopiclone, suriclone, zopiclone, 2-(7-chloro-2-naphthyl)-3-(5-methyl-2-oxohexyl)isoindolin-1-one, 2-(7-chloro-2-naphthyl)-3-(5-methyl-2-oxohexyl)isoindolin-1-yl-4-acetamidobutyrate, and 2-(7-chloro-2-naphthyl)-3-(5-methyl-5-hydroxy-2-oxohexyl)-1-isoindolin-1-yl-4-acetamidobutyrate.			
ST	GABA receptor modulators for stuttering treatment;			
IT	Drug delivery system for stuttering treatment			
IT	(GABA receptor modulators for alleviating stuttering)			
IT	GABA receptor modulators for alleviating stuttering			
IT	RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study, unclassified); DC (Process)			
IT	(GABA receptor modulators for alleviating stuttering)			
IT	GABA agonists for alleviating stuttering			
IT	RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study, unclassified); DC (Process)			
IT	(GABA agonists for alleviating stuttering)			

IT Brain, central nervous system (Gilman 1990); **GABA** receptor modulators for alleviating **stuttering**)

IT Drug delivery systems (buccal) **GABA** receptor modulators for alleviating **stuttering**

IT Protein class (RL: BAC; activity or effector, except adverse); THU (Therapeutic use); EIL (Biological study); USES (Uses) (diagnostic); **GABA** receptor modulators for alleviating **stuttering**)

IT Nervous system (disease) **GABA** receptor modulators for alleviating **stuttering**)

IT Drug delivery systems (epilepsy) **GABA** receptor modulators for alleviating **stuttering**

IT Drug delivery systems (infantile) **GABA** receptor modulators for alleviating **stuttering**

IT Drug delivery systems (infantile) **GABA** receptor modulators for alleviating **stuttering**

IT Drug delivery systems (infantile) **GABA** receptor modulators for alleviating **stuttering**

IT Drug delivery systems (infantile) **GABA** receptor modulators for alleviating **stuttering**

IT Drug delivery systems (infantile) **GABA** receptor modulators for alleviating **stuttering**

IT Drug delivery systems (infantile) **GABA** receptor modulators for alleviating **stuttering**

IT Behavior (motoric) **GABA** receptor modulators for alleviating **stuttering**)

IT Drug delivery systems (nasal) **GABA** receptor modulators for alleviating **stuttering**

IT Drug delivery systems (oral) **GABA** receptor modulators for alleviating **stuttering**

IT Drug delivery systems (parenteral) **GABA** receptor modulators for alleviating **stuttering**

IT Drug delivery systems (rectal) **GABA** receptor modulators for alleviating **stuttering**

IT Disease (speech) **GABA** receptor modulators for alleviating **stuttering**

IT Drug delivery systems (topical) **GABA** receptor modulators for alleviating **stuttering**

IT Drug delivery systems (vaginal) **GABA** receptor modulators for alleviating **stuttering**

IT 50-06- enantiomers, biological studies 50-06-6D, Phenobarbital, 53-43-0, Dehydroepiandrosterone 53-43-0D, 57-43-2, Amobarbital

57-43-2D, Amobarbital, enantiomers and metabolites 57-83-0, Progesterone, biological studies 57-83-0D, Progesterone, enantiomers and metabolites 58-25-3, Chlordiazepoxide 58-25-3D, Chlordiazepoxide, enantiomers and metabolites 76-73-3, Secobarbital 76-73-3D, Secobarbital, enantiomers and metabolites 76-74-4, Pentobarbitone 76-74-4D, Pentobarbitone, enantiomers and metabolites 76-75-5, Thiopental 76-75-5D, Thiopental, enantiomers and metabolites 77-02-1, Aprobarbital 77-02-1D, Aprobarbital, enantiomers and metabolites 77-26-9, Butalbital 77-26-9D, Butalbital, enantiomers and metabolites 115-38-8, Mephobarbital 115-38-8D, Mephobarbital, enantiomers and metabolites 125-40-6, Butabarbital 125-40-6D, Butabarbital, enantiomers and metabolites 145-13-1, Pregnenolone 145-13-1D, Pregnenolone, enantiomers and metabolites 151-83-7, Methohexital 151-83-7D, Methohexital, enantiomers and metabolites 439-14-5, Diazepam 439-14-5D, Diazepam, enantiomers and metabolites 485-49-4, Bicuculline 485-49-4D, Bicuculline, enantiomers and metabolites 516-54-1 516-54-1D, enantiomers and metabolites 516-55-2, Allopregnanolone 516-55-2D, Allopregnanolone, enantiomers and metabolites 567-03-3, Tetrahydrodeoxycorticosterone 567-03-3D, Tetrahydrodeoxycorticosterone, enantiomers and metabolites 604-75-1, Oxazepam 604-75-1D, Oxazepam, enantiomers and metabolites 846-49-1, Lorazepam 846-49-1D, Lorazepam, enantiomers and metabolites 846-50-4, Temazepam 846-50-4D, Temazepam, enantiomers and metabolites 1005-93-2, Etbicuphat 1005-93-2D, enantiomers and metabolites 1134-47-0, .+-.Baclofen 1134-47-0D, .+-.Baclofen, enantiomers and metabolites 1449-89-4, Mebicuphat 1449-89-4D, enantiomers and metabolites 1622-62-4, Flunitrazepam 1622-62-4D, Flunitrazepam, enantiomers and metabolites 2078-54-8, Propofol 2078-54-8D, Propofol, enantiomers and metabolites 2955-38-6, Prazepam 2955-38-6D, Prazepam, enantiomers and metabolites 3289-22-3, Flucybene 3289-22-3D, enantiomers and metabolites 4406-37-5, Pregnanolone 4406-37-5D, Pregnanolone, enantiomers and metabolites 17617-23-1, Flurazepam 17617-23-1D, Flurazepam, enantiomers and metabolites 17617-45-7, Picrotoxinin 17617-45-7D, Picrotoxinin, enantiomers and metabolites 21416-53-5, Picroton 21416-53-5D, Picroton, enantiomers and metabolites 23092-17-3, Halazepam 23092-17-3D, Halazepam, enantiomers and metabolites 23930-19-0 23930-19-0D, enantiomers and metabolites 28911-01-5, Triazolam 28911-01-5D, Triazolam, enantiomers and metabolites 28981-97-7, Alprazolam 28981-97-7D, Alprazolam, enantiomers and metabolites 29617-43-4 29617-43-4D, enantiomers and metabolites 29975-16-4, Estazolam 29975-16-4D, Estazolam, enantiomers and metabolites 33125-97-2, Etomidate 33125-97-2D, Etomidate, enantiomers and metabolites 34985-87-0, Chlorazepam 34985-87-0D, Chlorazepam, enantiomers and metabolites 36104-80-0, Camazepam 36104-80-0D, Camazepam, enantiomers and metabolites 36735-22-5, Quazepam 36735-22-5D, Quazepam, enantiomers and metabolites 43200-80-2, Zopiclone 43200-80-2D, Zopiclone, enantiomers and metabolites 51052-72-3, Isobicyphat 51052-72-3D, enantiomers and metabolites 51486-74-9, Propylbicyphat 51486-74-9D, enantiomers and metabolites 52463-83-9, Pinazepam 52463-83-9D, Pinazepam, enantiomers and metabolites 53813-83-5, Suriclone 53813-83-5D, Suriclone, enantiomers and metabolites 57109-90-7, Chlorazepate 57109-90-7D, Chlorazepate, enantiomers and metabolites 59467-70-8, Midazolam 59467-70-8D, Midazolam, enantiomers and metabolites 109370-34-5, Avermectin B 109370-34-5D, Avermectin B, enantiomers and metabolites 117705-18-7 117705-18-7D, enantiomers and metabolites 133737-32-3, Pagoclone 133737-32-3D, Pagoclone, enantiomers and metabolites 133737-48-1 133737-48-1D, enantiomers and metabolites 153046-19-6 153046-19-6D,

enantiomers and metabolites 224790-70-9, Cloflubicyne 224790-70-9D,
 Cloflubicyne, enantiomers and metabolites 224790-71-0, Etbicythionat
 224790-71-0D, Etbicythionat, enantiomers and metabolites
 RL: BAC (Biological Activity or effector, except adverse); THU
 (Therapeutic use); BIOL (Biological study); USES (Uses)
 (GABA receptor modulators for alleviating **stuttering**
)

L10 ANSWER 2 OF 45 TOXIT
 ACCESSION NUMBER: 2001:0118 TOXLIT
 DOCUMENT NUMBER: C1111-141757Z
 TITLE: Methods and compositions using **GABA** receptor
 modulators for alleviating **stuttering**.
 AUTHOR: Murphy JJ; D'orlando KJ
 SOURCE: (L. O.). PCT Int. Appl. PATENT NO. 018670 02/08/2001
 (Interneuron Pharmaceuticals, Inc.).
 COTED: PIXXD2.
 PUB. COUNTRY: UNITED STATES
 DOCUMENT TYPE: Patent
 FILE SEGMENT: C1111
 LANGUAGE: English
 OTHER SOURCE: C1111-141757
 ENTRY MONTH: 2001

AB Methods of treating **stuttering** include treating people with
 .gamma.-aminobutyric acid (**GABA**) receptor modulators, including
 cyclopyrrolones. A second active agent may be used with **GABA**
 receptor modulators. Active enantiomers, active metabolites, and
 pharmaceutically acceptable salts of **GABA** receptor modulators,
 including cyclopyrrolones, are acceptable components of the compns. The
 cyclopyrrolone class of modulators includes pagoclone, suriclone,
 zopiclone, 2-(7-chloro-2-naphthyridin-1,8-yl)-3-(5-methyl-2-
 oxohexyl)isoindolin-1-one,
 2-(7-chloro-2-naphthyridin-1,8-yl)isoindolin-1-
 yl-4-acetamidobutanoate, and
 2-(7-chloro-1,8-naphthyridin-2-yl)-3-(5-methyl-
 5-hydroxy-2-oxohexyl)-1-isoindolinone.
 TI Methods and compositions using **GABA** receptor modulators for
 alleviating **stuttering**.
 AB Methods of treating **stuttering** include treating people with
 .gamma.-aminobutyric acid (**GABA**) receptor modulators, including
 cyclopyrrolones. A second active agent may be used with **GABA**
 receptor modulators. Active enantiomers, active metabolites, and
 pharmaceutically acceptable salts of **GABA** receptor modulators,
 including cyclopyrrolones, are acceptable components of the compns. The
 cyclopyrrolone class of modulators includes pagoclone, suriclone,
 zopiclone, 2-(7-chloro-2-naphthyridin-1,8-yl)-3-(5-methyl-2-
 oxohexyl)isoindolin-1-one, . . .
 ST **GABA** receptor modulator for **stuttering** treatment;
 cyclopyrrolone compound **stuttering** treatment

L10 ANSWER 3 OF 45 FULL
 ACCESSION NUMBER: 01:4769 USPATFULL
 TITLE: Anidinylamino heterocycle compounds useful as
 alpha-2
 adrenoceptor agonists
 INVENTOR(S): Murphy, Thomas Lee, Norwich, NY, United States
 D'orlando, Sophie Eva, Maineville, OH, United States
 Todd, Raymond Todd, Pleasant Plain, OH, United States
 Eldon, Russell James, Fairfield, OH, United States
 Abel, William Lee, Hamilton, OH, United States

PATENT ASSIGNMENT(S): es, Jeffrey Joseph, Hamilton, OH, United States
e Procter & Gamble Company, Cincinnati, OH, United
ates (U.S. corporation)

	NUMBER	DATE
PATENT INFORMATION:	6172095	20010109
	9823591	19980604
APPLICATION INFO.:	1999-308790	19990809 (9)
	1997-US20550	19971121
		19990809 PCT 371 date
		19990809 PCT 102(e) date

	NUMBER	DATE
PRIORITY INFORMATION:	1996-31756	19961125 (60)
DOCUMENT TYPE:	Patent	
PRIMARY EXAMINER:	aman, D. Margaret	
LEGAL REPRESENTATIVE:	lberman, James C.; Roof, Carl J.	
NUMBER OF CLAIMS:		
EXEMPLARY CLAIM:		
LINE COUNT:	05	

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB This invention involves compounds having the structure (I) as described in the claims, and enantiomers, optical isomers, stereoisomers, diastereomers, tautomers, addition salts, biohydrolyzable amides and esters thereof, as well as pharmaceutical compositions comprising such novel compounds. The invention also relates to the use of such compounds for preventing or treating disorders modulated by alpha-2 adrenoceptors.

##S174#

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

DETD to respiratory challenges such as inhaled citric acid. (See, e.g., Callaway, G. R. King, "Effects of Inhaled .alpha.2-Adrenoceptor and GABA-subunit Receptor Agonists on Citric Acid-Induced Cough and Tidal Volume Changes in Guinea Pigs", European Journal of Pharmacology, Vol. 220 (1992), The effectiveness of other alpha-2 agonists in the management of neurologic disorders has been demonstrated, including attention-deficit hyperactive disorder and Tourette's syndrome (See, e.g., Chappell P., M. Riddle, L. Scalap, K. Finch, R. Schultz, A. Amsten, J. Leckman & D. Cohen, "Guaifenesin treatment of comorbid attention-deficit hyperactivity disorder and Tourette's syndrome: preliminary clinical experience", Journal of American Academy of Child and Adolescent Psychiatry, Vol. 34 (1995), pp. 1140-1146), cognitive disorders.

L10 ANSWER 0145 EMBASE COPYRIGHT 2001 ELSEVIER SCI. B.V.DUPLICATE 1
ACCESSION : 001 0362 EMBASE Alert (EMBAL)
TITLE: treatment in Tourette syndrome: A double-blind,-controlled, crossover trial.
AUTHOR: I.S.; Wendlandt J.; Krieger M.; Giuliano J.
CORPORATE : J. Singer, Johns Hopkins Hospital, Harvey 811, 600 N.

110 Street, Baltimore, MD 21287-8811, United States.
infor@jhmi.edu

SOURCE: ogy, (13 Mar 2001) 56/5 (599-604). Refs: 32.
NEURA ISSN: 0028-3878

PUB. COUNTRY: United States

DOCUMENT TYPE: Article

LANGUAGE: English

SUMMARY LANGUAGE: English

AB Object: To investigate the effectiveness of baclofen for the treatment of tic disorder in children with **Tourette** syndrome (TS). Background: Baclofen, which contains both .gamma.-aminobutyric acid (**GABA**) and pantoic acid moieties, was suggested in an open-label protocol to be an effective treatment for TS. This is a double-blind, placebo-controlled study to investigate this medication in children with TS. Ninety subjects received, in a randomized sequence, 4-week medication cycles of baclofen (20 mg three times daily) and placebo with

a 2-week placebo washout period between the cycles. Outcome measures included the Clinical Global Impression (CGI) scale, and the Yale Global Tic Severity Scale (YGTS), the latter including subscales for total tics and overall impairment. Measures were assessed at baseline and on days

28, 42, and 56 of the study. Results: Ten children (seven boys and three girls) with TS participated. Nine subjects completed the protocol; one dropped out for psychosocial reasons. No major side effects were reported. The mean change in CGI score (-0.9) after 4 weeks of baclofen treatment as compared with placebo treatment showed a

significant improvement (95% CI, -1.7 to -0.1; $p = 0.04$). All subjects showed some amelioration of total YGTS score during baclofen treatment. The mean change in total YGTS score (-14.7) approached significance (95% CI, -30.3

to 0.0). Examination of differences between baclofen and placebo

treatment expressed as a percent change from baseline showed that baclofen had a statistically significant effect on both outcome measures. Subscale scores on YGTS showed that the reduction in total tic scores was primarily due to a reduction in the impairment score rather than a decrease in the number of tics. Conclusions: Children with TS may benefit from

treatment with baclofen, although improvements may be related to factors other than tics. Further studies directly comparing baclofen against other tic-suppressing agents are recommended.

AB Object: To investigate the effectiveness of baclofen for the treatment of tic disorder in children with **Tourette** syndrome (TS). Background: Baclofen, which contains both .gamma.-aminobutyric acid (**GABA**) and pantoic acid moieties, was suggested in an open-label protocol to be an effective treatment for TS. This is a double-blind, . . .

L10 ANSWER: CAPLUS COPYRIGHT 2001 ACS DUPLICATE 2

ACCESSION NUMBER: 2000:688058 CAPLUS

DOCUMENT NUMBER: 133:261534

TITLE: Treatment of post-traumatic stress disorder, obsessive-compulsive disorder and related neuropsychiatric disorders

INVENTOR(S): Fogel, Barry S.

PATENT ASSIGNEE: Synchroneuron, Llc, USA

SOURCE: PCT Int. Appl., 58 pp.

CODEN: PIXXD2

DOCUMENT TYPE: Patent

LANGUAGE: English

FAMILY APPLICATION NUMBER: 1

PATENT INFORMATION:

PATENT	KIND	DATE	APPLICATION NO.	DATE
-----	-----	-----	-----	-----
WO 20000928	A2	20000928	WO 2000-US7119	20000317
WO 20001228	A3	20001228		
W. CH, CN, JP, MX, NZ				
R. CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL,				
PRIORITY A	:	US 1999-273036	19990319	
AB	The p	ation describes a novel treatment for neuropsychiatric		
	disor	ding anxiety disorders, mood disorders, psychotic		
	disor	form disorders, and neuropsychiatric symptoms resulting		
	from	orders. The treatment of the present invention utilizes		
	any a	multaneously act as NMDA-type glutamate receptor		
	antago	GABA-A receptor agonists. Preferably these two		
activities				
	are c	ic of a single agent, for example acamprosate (calcium		
	N-ace	inate). Alternatively, sep. agents having these		
	activ	e combined as a compd. or mixt. and thereby administered		
	toget	vention also provides for a third agent that acts as a		
	non-c	MDA-receptor blocking agent or ion channel blocker, that		
	augme	ect of the primary treatment. A particularly preferred		
	ion c	king agent is magnesium.		
IT	Brain,			
	(G	Tourette syndrome; post-traumatic stress		
	di	essive-compulsive disorder and related neuropsychiatric		
	di	atment with NMDA glutamate antagonists and GABA		
A				
L10 ANSWER	OF	USPATFULL		
ACCESSION	NUMBER:	2000:171042	USPATFULL	
TITLE:		2-imidazolinyllaminoindole compounds useful as alpha-2		
INVENTOR(S)		Henry, Raymond Todd, Pleasant Plain, OH, United States		
		Sheldon, Russell James, Fairfield, OH, United States		
		Seibel, William Lee, Hamilton, OH, United States		
PATENT ASS	SEE(S)	The Procter & Gamble Company, Cincinnati, OH, United		
		States (U.S. corporation)		
		NUMBER	DATE	
		-----	-----	
PATENT INFO	MATION	US 6162818	20001219	
APPLICATION	INFO.:	US 1999-290731	19990413 (9)	
RELATED AP	N. INF	Continuation of Ser. No. WO 1997-US20801, filed on 21		
		Nov 1997		
		NUMBER	DATE	
		-----	-----	
PRIORITY II	ORMATI	US 1996-31777	19961111 (60)	
DOCUMENT T	E:	Utility		
PRIMARY EX	INER:	McKane, Joseph K.		
ASSISTANT	AMINER:	Oswecki, Jane C.		
LEGAL REPR	ENTATI	Bott, Cynthia M.; Kellerman, James C.; Clark, Karen F.		
NUMBER OF	AIMS:	42		
EXEMPLARY	AIM:	1		
LINE COUNT:		2524		
CAS INDEX	IS AV	ABLE FOR THIS PATENT.		
AB	This invent	involves compounds having the following structure:		
	##S	l## where: a) R.sub.1 is hydrogen; or alkyl; bond (a) is a		
single				
	or	double bond;		

b) F.sub.2 and R.sub.3 are each independently selected from hydrogen; unsubstituted C.sub.1 -C.sub.3 alkanyl, alkenyl or alkynyl; cycloalkanyl, cycloalkenyl; unsubstituted C.sub.1 -C.sub.3 alkylthio or alkoxy; hydroxy; thio; nitro; cyano; amino; C.sub.1 -C.sub.3 alkylamino or C.sub.1 -C.sub.3 dialkylamino and halo;

c) F.sub.4, R.sub.5 and R.sub.6 are each independently selected from hydrogen; unsubstituted C.sub.1 -C.sub.3 alkanyl, alkenyl or alkynyl; cycloalkanyl, cycloalkenyl; unsubstituted C.sub.1 -C.sub.3 alkylthio or alkoxy; hydroxy; thio; nitro; cyano; amino; C.sub.1 -C.sub.3 alkylamino or C.sub.1 -C.sub.3 dialkylamino; halo; and 2-imidazolinylamino; and wherein one and only one of R.sub.4, R.sub.5 and R.sub.6 is 2-imidazolinylamino;

d) R.sub.7 is selected from hydrogen; unsubstituted C.sub.1 -C.sub.3 alkanyl, alkenyl or alkynyl; cycloalkanyl, cycloalkenyl; unsubstituted C.sub.1 -C.sub.3 alkylthio or alkoxy; hydroxy; thio; nitro; cyano; amino; C.sub.1 -C.sub.3 alkylamino or C.sub.1 -C.sub.3 dialkylamino and halo;

e) the compound is not 4-(2-imidazolinylamino)indole;

enantiomers, optical isomers, stereoisomers, diastereomers, tautomers, addition salts, biohydrolyzable amides and esters thereof, and pharmaceutical compositions comprising such novel compounds. The invention also relates to the use of such compounds for treating disorders mediated by alpha-2 adrenoceptors.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

DETD to respiratory challenges such as inhaled citric acid. (See, e.g., Callaway, J. & R. King, "Effects of Inhaled .alpha.2-Adrenoceptor and GABA.sub.B Receptor Agonists on Citric Acid-Induced Cough and Tidal Volume Changes in Guinea Pigs", European Journal of Pharmacology, Vol. 220 (1992), The effectiveness of other alpha-2 agonists in the management of neurologic disorders has been demonstrated, including attention-deficit hyperactive disorder and Tourette's syndrome (See, e.g., Chappell P., M. Riddle, L. Scamill, K. Lynch, R. Schultz, A. Arnsten, J. Leckman & D. Cohen, "Guanfacine treatment of comorbid attention-deficit hyperactivity disorder and Tourette's syndrome: preliminary clinical experience", Journal of American Academy of Child and Adolescent Psychiatry, Vol. 34 (1995), pp. 1140-1146), cognitive disorders. . . .

L10 ANSWER 7 OF 45 USPATFULL

ACCESSION NUMBER: 2000:137814 USPATFULL
TITLE: Allelic polygene diagnosis of reward deficiency syndrome and treatment
INVENTOR(S): Blum, Kenneth, San Antonio, TX, United States
PATENT ASSIGNEE(S): City of Hope National Medical Center, Duarte, CA, United States (U.S. corporation)
The University of Texas System AMD Board of Regents, Austin, TX, United States (U.S. corporation)

	NUMBER	DATE
PATENT INFORMATION:	US 6132724	20001017
APPLICATION INFO.:	US 1998-69886	19980429 (9)
DOCUMENT TYPE:	Utility	

PRIMARY EXAMINER: Witz, Jean C.
LEGAL REPRESENTATIVE: Hodgins, Daniel S.
NUMBER OF CLAIMS: 9
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 8 Drawing Figure(s); 8 Drawing Page(s)
LINE COUNT: 20845

AB Enhancement of attentional processing is attained by administration of an endorphinase inhibitor or enkephalinase inhibitor and optionally, a dopamine precursor, or a serotonin precursor, a GABA precursor, or an endorphin or enkephalinase releaser, or certain herbal compounds including Rhodiola rosea extract (Pharmaline) and/or Huperzine. These components promote restoration of normal neurotransmitter function and the components combined enhance the release of dopamine at the nucleus accumbens and are non-addictive. Use of the dopamine precursors L-phenylalanine, or L-Tyrosine, the enkephalinase inhibitor D-phenylalanine, and/or the serotonin precursor -hydroxytryptophan and

a natural acetylcholinesterase inhibitor and chromium salts (i.e. picolinate, nicotinate, etc.) is especially preferred, but not limited to assist in relieving symptoms associated with brain phenylalanine deficiency.

DETD a number of receptors are involved in various types of substance abuse including dopamine, serotonin, cannabinoid, nitric oxide, nicotinic muscarinic, GABA, and others. The diagnosis of Tourette's syndrome is dependent upon the presence of motor tics, and dopamine plays a major role in the regulation of muscle. .

L10 ANSWER 8 OF 45 USPATFULL

ACCESSION NUMBER: 2000:121514 USPATFULL
TITLE: 6-(2-imidazolinyllamino)quinoxaline compounds useful as alpha-2 adrenoceptor agonists
INVENTOR(S): Maurer, Peter J., Cincinnati, OH, United States
Henry, Raymond T., Pleasant Plain, OH, United States
Sheldon, Russell James, Fairfield, OH, United States
PATENT ASSIGNEE(S): The Procter & Gamble Company, Cincinnati, OH, United States (U.S. corporation)

	NUMBER	DATE
PATENT INFORMATION:	US 6117871	20000912
APPLICATION INFO.:	US 1996-755941	19961125 (8)
RELATED APPLN. INFO.:	Continuation-in-part of Ser. No. US 1995-496707, filed on 29 Jun 1995, now abandoned which is a continuation-in-part of Ser. No. US 1993-169785, filed on 17 Dec 1993, now abandoned	
DOCUMENT TYPE:	Utility	
PRIMARY EXAMINER:	Fay, Zohreh	
LEGAL REPRESENTATIVE:	Bott, Cynthia M.; Kellerman, James C.; Suter, David L.	
NUMBER OF CLAIMS:	18	
EXEMPLARY CLAIM:	1	
LINE COUNT:	1432	

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB The subject invention relates to methods of treating alpha-2 adrenoceptor modulated disorders, comprising administration, to a mammal in need of such treatment, of a safe and effective amount of a compound having the following structure: ##STR1## wherein: (a) R is unsubstituted C.sub.1 -C.sub.3 alkanyl or alkenyl; and

(b) is selected from hydrogen; unsubstituted C.sub.1 -C.sub.3 alkanyl or phenyl; unsubstituted C.sub.1 -C.sub.3 alkylthio or alkoxy; hydroxy; thio; and halo.

The subject invention also relates compounds and compositions for preventing or treating of disorders modulated by alpha-2 adrenoceptors.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

DETD . . . to respiratory challenges such as inhaled citric acid. (See, e.g., Callaway, J. & R. King, "Effects of Inhaled .alpha.2-Adrenoceptor and GABA.sub.3 Receptor Agonists on Citric Acid-Induced Cough and Tidal Volume Changes in Guinea Pigs", European Journal of Pharmacology Vol. 220 (1992), . . . The effectiveness of other alpha-2 agonists in the management of neurologic disorders has been demonstrated, including attention-deficit hyperactive disorder and Tourette's syndrome (See, e.g., Chappell P., M. Riddle, L. Scarff, K. Lynch, R. Schultz, A. Arnsten, J. Leckman & D. Cohen, "Guanfacine treatment of comorbid attention-deficit hyperactivity disorder and Tourette's syndrome: preliminary clinical experience", Journal of American Academy of Child and Adolescent Psychiatry, Vol. 34 (1995), pp. 1140-1146), cognitive disorders. . .

L10 ANSWER OF . . . USPATFULL

ACCESSION NUMBER: 2000:113979 USPATFULL

TITLE: 2-imidazolinyaminobenzoxazole compounds useful as alpha-2 adrenoceptor agonists

INVENTOR(S): Henry, Raymond Todd, Pleasant Plain, OH, United States
Sheldon, Russell James, Fairfield, OH, United States
Seibel, William Lee, Hamilton, OH, United States

PATENT ASSIGNEE(S): The Procter & Gamble Company, Cincinnati, OH, United States (U.S. corporation)

	NUMBER	DATE
PATENT INFORMATION:	US 6110952	20000829
	WO 9823611	19980604
APPLICATION INFO.:	US 1999-308792	19990809 (9)
	WO 1997-US20803	19971121
		19990809 PCT 371 date
		19990809 PCT 102(e) date

	NUMBER	DATE
PRIORITY INFORMATION:	US 1996-31787	19961125 (60)
DOCUMENT TYPE:	Utility	
PRIMARY EXAMINER:	McKane, Joseph	
ASSISTANT EXAMINER:	Wright, Sonya N	
LEGAL REPRESENTATIVE:	Kellerman, James C.; Roof, Carl J.	
NUMBER OF CLAIMS:	42	
EXEMPLARY CLAIM:	1	
LINE COUNT:	1879	

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB This invention relates to compounds of formula I, (2-imidazolinyamino)benzoxazoles. The compounds have been found to be alpha-2 adrenoceptor agonists and are useful for treatment of disorders modulated by alpha-2 adrenoceptors.

CAS INDEXED AVAILABLE FOR THIS PATENT.

DETD respiratory challenges such as inhaled citric acid. (See, e.g., Callahan, J. & R. King, "Effects of Inhaled .alpha.2-Adrenoceptor and .alpha.2-Adrenoceptor Agonists on Citric Acid-Induced Cough and Airway Changes in Guinea Pigs", European Journal of Pharmacology, Vol. 220 (1992), The effectiveness of other alpha-2 agonists in the management of neurologic disorders has been demonstrated, including attention-deficit hyperactive disorder and Tourette's syndrome (See, e.g., Chappell P., M. Riddle, L. Scahill, R. Schultz, A. Arnsten, J. Leckman & D. Cohen, "Guinea Pig Model of comorbid attention-deficit hyperactivity disorder and Tourette's syndrome: preliminary clinical experience", Journal of American Academy of Child and Adolescent Psychiatry, Vol. 34 (1995), pp. 1140-1146), cognitive disorders.

L10 ANSWER OF USPATFULL

ACCESSION NO. 2000:54156 USPATFULL

TITLE: Methods of treating tardive dyskinesia and other movement disorders using NMDA receptor antagonists

INVENTOR(S) Fogel, Barry S., Waban, MA, United States
PATENT ASSIGNEE(S) Synchroneuron, LLC, Waban, MA, United States (U.S. corporation)

	NUMBER	DATE
PATENT INFO	US 6057373	20000502
APPLICATION NO.	US 1999-224829	19990104 (9)
RELATED APPL.	Continuation-in-part of Ser. No. US 1997-861801, filed on 22 May 1997, now patented, Pat. No. US 5866585	
DOCUMENT TYPE	Utility	
PRIMARY EXAMINER	MacMillan, Keith D.	
ASSISTANT EXAMINER	Kim, Vickie Y.	
LEGAL REPRESENTATIVE	Choate, Hall & Stewart; Pasternack, Sam	
NUMBER OF PAGES	52	
EXEMPLARY	1	
LINE COUNT	1728	

CAS INDEXED AVAILABLE FOR THIS PATENT.

AB The present invention describes a novel treatment for movement disorders, including tardive dyskinesia and tardive dystonia, and focal dyskinesias due to neuroleptics, including blepharospasm, Meige syndrome, and occupational dystonias. The treatment of the present invention utilizes agents that act as NMDA-type glutamate receptor antagonists. The invention also involves the use of an ion channel blocking agent to augment the therapeutic action of the drug treatments described. A particularly preferred ion channel blocking agent is magnesium.

CAS INDEXED AVAILABLE FOR THIS PATENT.

SUMMARY And neuroleptic treatment, clonazepam, a benzodiazepine with GABA-A agonist actions, has some efficacy in the treatment of Tourette's syndrome (Steingard et al., J. Am Acad Child Psychiatry, March-April, 33:394-9, 1994). Sedation and ataxia are the dosage of.

DETD As, GABA-A agonists alone are not particularly potent inhibitors of tics. Therefore, NMDA antagonism is likely a necessary component of the therapeutic. of memantine is more important

than the agonism. Evidence disclosed herein suggests that

mo: ers, such as tics and **Tourette's**, will respond
 in: lar to tardive movement disorders to drug therapies
 ha: gonist activity.

L10 ANSW: EPIDS COPYRIGHT 2001 DERWENT INFORMATION LTD
 ACCESSION 001-071285 [08] WPIDS
 DOC. NO. (2001-019987
 TITLE: Identifying genes of interest and relevance to
 neurological disorders or dysfunctions such as
 Parkinson's disease involves use of biological array
 including all genes associated with neurotransmitter
 molecules.
 DERWENT C: 04 D16
 INVENTOR(S) REEK, M J; LAFORGE, K S; SPANGLER, R
 PATENT AS. UYRQ) UNIV ROCKEFELLER
 COUNTRY C: 3
 PATENT IN: IC

PATEL	ATE	WEEK	LA	PG
WO 2000-01221	(200108)*	EN	76	
RE	DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW MZ			
	SE SL SZ TZ UG ZW			
	AT AU AZ BA BB BG BR BY CA CH CN CR CU CZ DE DK DM DZ			
	ES GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK			
	LV MA MD MG MK MN MW MX MZ NO NZ PL PT RO RU SD SE SG			
	TM TR TT TZ UA UG US UZ VN YU ZA ZW			

APPLICATION AI

PATEL	APPLICATION	DATE
WO 2000-01221	WO 2000-US16706	20000616

PRIORITY A: S 1999-334113 19990616
 AN 2001- WPIDS
 AB WO 2000-01221 B: 20010207
 NOVEL ing genetic predisposition to, susceptibility to
 devel characteristics of, or persistence of physiological or
 path se to, a neurotransmitter factor-related condition
 (NFC Identifying genetic polymorphisms in neurotransmitter
 genes ith NFC, using a multiple biological sample array, is
 new.
 CLAIM RIPTION - An INDEPENDENT CLAIM is also included for
 makin logical chip plate (I), comprising attaching a wafer
 comp surface, a set of probe arrays (II) each comprising a
 coll es, at least two of which, are different, and arranged
 in s ed and physically addressable manner, to a body
 comp f defined spaces. (II) is selected from a family of
 neur enes known to be affected by exposure to addictive
 agent hol. The probes are selected from a family of
 neuro enes known to be involved in a neurological disorder or
 dysfu se to strain, stress, gastrointestinal function,
 immune
 func live function or signal transduction. The method
 comp a material resistant to the flow of a liquid, to
 surr arrays, creating test wells.
 iden ing a biological chip plate which is useful for
 c predisposition to, susceptibility to development of,

characterized by, or persistence of physiological or pathological
 response to genetic polymorphisms or gene expression in several
 neurological disorders, preferably opioid system genes, associated with
 the disorders. The arrays are useful in prognosis of neurological
 disorders such as schizophrenia, Tourette syndrome, drug abuse,
 attention deficit disorder, anxiety, depression obsessive-compulsive disorder,
 stroke, response to pain, hypertension, vascular disorders,
 migraines, Alzheimer's disease, aggressive behavior, premenstrual
 syndrome, neuropathy, suppression of alcohol intake, and
 Parkinson's disease. (All claimed). A DNA array can determine the
 presence of a pathogenic organism based on characteristic DNA
 sequences. The array provides a multifunction analytical
 capability as in the study of RNA abnormalities or polymorphisms, and
 part of the nucleotide polymorphisms. It will yield quantitative
 information on physiological and/or pathological condition of the
 test subject. The analysis of the DNA of the subject will provide
 information on the subject genotype and corresponding genetic
 predisposition. The methods allow many tests to be set up and processed
 together. They allow much higher throughput of test samples,
 these methods improve the efficiency of performing assays on biological
 chips. The biological and genetic conditions can be identified using
 the arrays. This is rapid and inexpensive.
 Dwg.

TECH.

adrenoreceptor, DARPP-32, dopamine D1 receptor, dopamine D2 receptor,
 and acetylcholine receptor genes such as α -E and subtypes,
 GABA (gamma-aminobutyric acid) receptor (muscarinic) genes,
 glutamate receptor genes, and NMDA (undefined) receptor genes and the
 genes are associated with. . . . as opiate, cocaine,
 neuroendocrine, addiction. The neurotransmitter genes are involved in a
 hereditary disorder such as addiction, schizophrenia, **Tourette**
 syndrome, attention deficit disorder, anxiety, depression
 obsessive-compulsive disorder, stroke, obesity, response to pain,
 hyperlipidemia, vascular disorders, migraine, nausea,

L10 ANSWER 2
 ACCESSION NUMBER 000-411597 [35] WPIDS
 CROSS REFERENCE 009-444313 [37]
 DOC. NO. 000-124615
 TITLE: Treating movement disorders using agents which increase
 ABA-A neurotransmission and decrease NMDA-glutamate
 neurotransmission.
 DERWENT CLASSIFICATION B06
 INVENTOR(S) GEL, B S
 PATENT AGENT (S) (INC-N) SYNCHRONEURON LLC
 COUNTRY OF ORIGIN
 PATENT IN STATUS

PATENT	FILE	WEEK	LA	PG
WO 2000/0525	200035	*	EN	61
E DK ES FI FR GB GR IE IT LU MC NL PT SE				
P MX NZ				
AU 2000/00605	200042			

APPLICATION

PATENT NO.	APPLICATION	DATE
WO 2000-017343	WO 1999-US27343	19991118
AU 2000-17347	AU 2000-17347	19991118

FILING DATE

PATENT NO.	PATENT NO.
AU 2000-17347	WO 200028999

PRIORITY NO. 1998-193892 19981118

AN 2000-017343 EPIDS

CR 1999-017343

AB WO 2000-017343 B: 20000905

NOVE 1999-017343 for treating movement disorders comprises

administered

an agent which decreases GABA-A neurotransmission and decreases neurotransmission to a patient with a movement disorder. DESCRIPTION - INDEPENDENT CLAIMS are included for: for treating movement disorders which comprises agent (I) that acts as a GABA-A receptor agonist and a which acts as a NMDA-type glutamate receptor antagonist and them to the patient; for assessing risk of developing a neuroleptic or blocker-induced movement disorder which comprises and tests of total body magnesium status; for preventing neuroleptic or dopamine receptor movement disorders and for treating movement disorders which administering magnesium ions; and methods/compositions based upon various combinations of the

anti-dyskinesia. ACTION - NMDA Receptor antagonist; GABA-A agonist. to treat simple or multiple tics, **Tourette's** dystonias, blepharospasm and Meige syndrome. The

synd disorders are related to **GABA** deficiency in the basal ganglia or to excitotoxicity (all claimed). The method may be used

to treat dyskinesia and movement disorders induced by exposure to neuroleptic (psychotic) drugs.

Dwg.

AB

- An agent which decreases GABA-A neurotransmission and decreases neurotransmission to a patient with a movement disorder. ACTION - NMDA Receptor antagonist; GABA-A agonist. to treat simple or multiple tics, **Tourette's** dystonias, blepharospasm and Meige syndrome. The

synd disorders are related to **GABA** deficiency in the basal ganglia or to excitotoxicity (all claimed). The method may be used

used to treat dyskinesia.

L10 ANSWER PLEASE COPYRIGHT 2001 ELSEVIER SCI. B.V.

ACCESSION NO. 04103 EMBASE

TITLE: GABA deficiency syndrome: A biogenetic model for the

diagnosis and treatment of impulsive, addictive, and
 impulsive behaviors.
 AUTHOR: Miller D.; Braverman E.R.; Holder J.M.; Lubar J.F.; Monastera
 CORPORATE: Miller D.; Lubar J.O.; Chen T.J.H.; Comings D.E.
 SOURCE: Blum, Department of Biological Sciences, University
 North Texas, Denton, TX, United States
 Journal of Psychoactive Drugs, (2000) 32/SUPPL. (1-112).
 638
 ISSN: 0279-1072 CODEN: JPDRD3
 COUNTRY: United States
 DOCUMENT: General Review
 FILE SEGM: Human Genetics
 Clinical Biochemistry
 Psychiatry
 Drug Literature Index
 Drug Dependence, Alcohol Abuse and Alcoholism
 LANGUAGE: English
 SUMMARY L: English
 AB The system, and in particular the dopamine D(2) receptor,
 has been
 inter the mesolimbic brain region induces 'reward' when dopamine
 (DA) from the neuron at the nucleus accumbens and interacts
 with the D(2) receptor. 'The reward cascade' involves the release
 of serotonin at the hypothalamus stimulates enkephalin,
 which inhibits GABA at the substantia nigra, which in
 turn increases the amount of DA released at the nucleus accumbens or
 'reward'. It is well known that under normal conditions in the
 reward system, dopamine maintain our normal drives. In fact, DA has become to be
 known as the 'pleasure molecule' and/or the 'antistress molecule.' When
 DA is released at the synapse, it stimulates a number of DA receptors
 (D1- D5). The literature suggests that when there is a
 dysfunction in the reward cascade, which could be caused by certain
 genetic factors (e.g., dopamine deficiency), especially in the DA system causing a
 hypo-dopamine state, the brain of that person requires a DA fix to
 feel good. This leads to multiple drug-seeking behavior. This is so
 because cocaine, heroin, marijuana, nicotine, and glucose all
 cause neuronal release of brain DA, which could heal the
 abnormality. Certainly after ten years of study we could say with
 confidence that carriers of the DAD2 receptor A1 allele have compromised
 D(2) receptors. The lack of D2 receptors causes individuals to have
 a high risk for severe addictive, impulsive and compulsive behavioral
 problems such as severe alcoholism, cocaine, heroin, marijuana and
 nicotine addiction, bingeing, pathological gambling, sex addiction,
 ADHD, depression, anxiety, autism, chronic violence,
 post-traumatic stress disorder, schizoid/avoidant cluster, conduct
 disorder, and
 case of multiple genes and environmental stimuli
 (plasticity) that cause aberrant behaviors, Blum united this
 hypothesis under the rubric of a reward deficiency syndrome.
 AB The 'reward cascade' involves the release of serotonin, which in
 turn stimulates enkephalin, which in turn inhibits
 GABA at the substantia nigra, which in turn fine tunes the amount
 of DA released at the nucleus accumbens or 'reward'. . . compulsive
 behaviors, such as severe alcoholism, cocaine, heroin,

mar: ... glucose bingeing, pathological gambling, sex
add: ... Syndrome, autism, chronic violence,
post: ... disorder, schizoid/avoidant cluster, conduct
disorder
and ... In order to explain the. . .

L10 ANS: ; COPYRIGHT 2001 BIOSIS

ACCESSION: 76 BIOSIS

DOCUMENT: 00054076

TITLE: ... deficiency syndrome: A biogenetic model for the
and treatment of impulsive, addictive, and
behaviors.

AUTHOR(S): ... (1); Braverman, Eric R.; Holder, Jay M.;
... F.; Monastra, Vincent J.; Miller, David;

Lubar,

...; Chen, Thomas J. H.; Comings, David E.

CORPORATE: ... Department of Biological Sciences, University of North
... TX USA

SOURCE: ... Psychoactive Drugs, (November, 2000) Vol. 32,
... pp. i-iv, 1-112. print.
...-1072.

DOCUMENT: ... view

LANGUAGE:

SUMMARY I

AB The ... and in particular the dopamine D2 receptor, has
been ... mechanisms. The net effect of neurotransmitter
into ... brain region induces "reward" when dopamine
(DA) ... neuron at the nucleus accumbens and interacts
with ... "The reward cascade" involves the release of
sero ... at the hypothalamus stimulates enkephalin, which
in ... the substantia nigra, which in turn fine
tune ... released at the nucleus accumbens or "reward
site."

It ... under normal conditions in the reward site DA works
to ...elves. In fact, DA has become to be known as the
"ple ... the "antistress molecule." When DA is released
into ... stimulates a number a DA receptors (D1-D5) which
resu ... of well-being and stress reduction. A
cons ... suggests that when there is a dysfunction in
the ... , which could be caused by certain genetic
vari ... specially in the DA system causing a
hypo ... the brain of that person requires a DA fix to

feel

good ... multiple drug-seeking behavior. This is so
beca ... heroin, marijuana, nicotine, and glucose all
caus ... onal release of brain DA, which could heal the
abno ... only after ten years of study we could say with
cont ... of the DAD2 receptor A1 allele have compromised
D2 ... lack of D2 receptors causes individuals to have a
hig: ... dictive, impulsive and compulsive behavioral
pro: ... ere alcoholism, cocaine, heroin, marijuana and
nicc ... geing, pathological gambling, sex addiction,
ADH: ... , autism, chronic violence,
post ... der, schizoid/avoidant cluster, conduct

disorder

and

cas ... In order to explain the breakdown of the reward
(pl. ... ple genes and environmental stimuli
hyp ... ant aberrant behaviors, Blum united this
der the rubric of a reward deficiency syndrome.

AB. ... " involves the release of serotonin, which in

tur: stimulates enkephalin, which in turn inhibits
 GAB: gra, which in turn fine tunes the amount
 of: nucleus accumbens or "reward. . . compulsive
 beha: such as severe alcoholism, cocaine, heroin,
 mar: se, glucose bingeing, pathological gambling, sex
 add: e's Syndrome, autism, chronic violence,
 post: order, schizoid/avoidant cluster, conduct
 disorder
 and In order to explain the breakdown. . .

L10 ANS COPYRIGHT 2001 ACS DUPLICATE 3
 ACCESSIO: 464180 CAPLUS
 DOCUMENT: 111440
 TITLE: ds using agents simultaneously acting as
 -type glutamate receptor antagonists and GABA-A
 ptor agonists for treating tardive dyskinesia and
 r movement disorders
 INVENTOR: l, Barry S.
 PATENT A: throneuron, LLC, USA
 SOURCE: Int. Appl., 106 pp.
 I: PIXXD2
 DOCUMENT: nt
 LANGUAGE: sh
 FAMILY A:
 PATENT IN: I

PATI	ATE	APPLICATION NO.	DATE
WO	9990722	WO 1999-US144	19990113
WO	9991202		
	JP, MX, NZ		
	DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL,		
US	9990914	US 1998-6641	19980113
US	0000502	US 1999-224829	19990104
AU	9990802	AU 1999-21041	19990113
EP	0001102	EP 1999-901314	19990113
	DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT,		
WO	0000525	WO 1999-US27343	19991118
WO	0000720		
	JP, MX, NZ		
	DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL,		
	SE		
PRIORITY	IFO.	US 1998-6641	19980113
		US 1998-193892	19981118
		US 1999-224829	19990104
		US 1997-861801	19970522
		WO 1999-US144	19990113
AB	The	treatments for movement disorders, including	
	tard	tardive dyskinesia, tic disorders, Tourette's	
	sync	and other focal dystonias. The treatments use	
	agen	ly act as NMDA-type glutamate receptor	
	anta	e treatments of the invention use agents that	
	sim	DA-type glutamate receptor antagonists and GABA-A	
	rec	erably, these two activities are characteristic	
of			
	a s	prostate. Alternatively, sep. agents having	
these			
	acti	ed and administered together. The invention also	

providing a third agent that can be used in combination with a treatment for movement disorders that acts as a non-competitive NMDA-receptor blocker or ion channel blocker that augments the effect of the primary treatment. A particularly preferred ion channel blocking agent is

magnesium. Alternatively, magnesium can be administered alone for the prevention and treatment of movement disorders.

IT Brain disease syndrome; agents simultaneously acting as NMDA-type glutamate receptor antagonists and GABA-A receptor agonists (for treatment of movement disorders)

L10 ANSWER 1 OF 45

ACCESSION NUMBER: 19930103 TOXLIT

DOCUMENT NUMBER: CA 1440M

TITLE: Methods for combining agents simultaneously acting as NMDA-type glutamate receptor antagonists and GABA-A receptor

agonists

for the treatment of tardive dyskinesia and other movement disorders.

AUTHOR: Foster, J.

SOURCE: (1) U.S. Pat. Int. Appl. PATENT NO. 9936064 07/22/1999 (S. Kure, LLC).

CO. 1440M

PUB. COUNTRY: UNITED STATES

DOCUMENT NUMBER: Pat.

FILE SEQUENCE: CA

LANGUAGE: English

OTHER SOURCE: CA 1440

ENTRY MONTH: 1993

AB The invention describes treatments for movement disorders, including tardive dyskinesia, tardive dystonia, tic disorders, **Tourette** syndrome, blepharospasm, and other focal dystonias. The treatments use agents that simultaneously act as NMDA-type glutamate receptor antagonists. Additionally, treatments of the invention use agents that simultaneously act as GABA-type glutamate receptor antagonists and GABA-A receptor agonists. Preferably, these two activities are characteristic of a single agent, e.g. acamprosate. Alternatively, separate agents having these activities can be combined and administered together. The invention also includes a third agent that can be used in combination with a treatment for movement disorders, that acts as a non-competitive NMDA-receptor blocker or ion channel blocker that augments the effect of the primary treatment. A particularly preferred ion channel blocker agent is magnesium. Alternatively, magnesium can be administered alone for the prevention and treatment of movement disorders.

AB The invention describes treatments for movement disorders, including tardive dyskinesia, tardive dystonia, tic disorders, **Tourette** syndrome, blepharospasm, and other focal dystonias. The treatments use agents that simultaneously act as NMDA-type glutamate receptor antagonists. Additionally, treatments of the invention use agents that simultaneously act as GABA-type glutamate receptor antagonists and GABA-A receptor agonists. Preferably, these two activities are characteristic of a single agent, e.g. acamprosate. Alternatively, separate agents having these activities can be combined and administered together.

L10 ANSWER 7 OF 45

ACCESSION NUMBER: 19931 USPATFULL

TITLE: 2,6-dimethylamino heterocyclic compounds useful as dopamine receptor agonists

INVENTOR: Peter J., Cincinnati, OH, United States

Jeffrey J., Hamilton, OH, United States
 William L., Hamilton, OH, United States
 Daniel P., Bloomington, IN, United States
 Russell James, Fairfield, OH, United States
 Raymond T., Pleasant Plain, OH, United States
 PATENT ATTORNEY(S): Foster & Gamble Company, Cincinnati, OH, United States
 (U.S. corporation)

	NUMBER	DATE
PATENT INFORMATION:	1995	19991012
APPLICATION INFO.:	55085	19961125 (8)
RELATED P. INFO.:	Continuation-in-part of Ser. No. US 1995-478708, filed Jul 1995, now patented, Pat. No. US 5663189 which is a continuation-in-part of Ser. No. US 1993-86482, filed Jul 1993, now abandoned	

DOCUMENT TYPE: Y
 PRIMARY INVENTOR: Lee, Jerome D.
 LEGAL REPRESENTATIVE: Mann, James C.; Roof, Carl J.; Suter, David L.
 NUMBER OF CLAIMS:
 EXEMPLAR CLAIM:
 LINE COUNT:

CAS INDEX IS AVAILABLE FOR THIS PATENT.
 ABSTRACT: The subject invention relates to compounds having the structure:
 #1## wherein n is an integer from 1 to about 3;

with (1) and Y are independently selected from O, S and CH.sub.2,
 and at least one of X and Z being O or S;

(2) is unsubstituted straight or branched chain alkanyl or alkanoxy
 having from 1 to 6 non-hydrogen atoms; and

(3) is selected from hydrogen, methyl, cyano, and halo;

pharmaceutical compositions containing such compounds; and the use of
 such compounds for the prevention or treatment of disorders modulated by
 alpha-2 adrenoceptors.

CAS INDEX IS AVAILABLE FOR THIS PATENT.
 DETD: . . . to respiratory challenges such as inhaled citric acid. (See,
 e.g., Callaway, J. . . , "Effects of Inhaled .alpha.2-Adrenoceptor
 agonists on Citric Acid-Induced Cough
 and Tidal Volume in Guinea Pigs", European Journal of
 Pharmacology, Vol. . . . (1992), . . . The effectiveness of other
 alpha-2 agonists in the management of neurologic disorders has been
 demonstrated, including attention-deficit hyperactive disorder and
 Tourette's syndrome (e.g., Chappell P., M. Riddle, L.
 Schwartz, K. Lynch, . . . , Schultz, A. Arnsten, J. Leckman & D. Cohen,
 "Clonidine treatment of comorbid attention-deficit hyperactivity
 disorder and Tourette's syndrome: preliminary clinical
 evidence", Journal of the American Academy of Child and Adolescent
 Psychiatry, Vol. . . . , pp. 1140-1146), cognitive disorders. . .

L10 ANSWER: 3 OF 45 US
 ACCESSION NUMBER: 92 USPATFULL
 TITLE: (azolinylamino)quinoline compounds useful as
 adrenoceptor agonists
 INVENTOR: Thomas Lee, Oxford, OH, United States

PATENT ASSIGNOR(S): Wyeth & Gamble Company, Cincinnati, OH, United States corporation)

	NUMBER	DATE
PATENT INFORMATION:	412	19990622
APPLICATION INFO.:	8-159698	19980924 (9)
RELATED P. INFO.:	Continuation of Ser. No. US 1996-756085, filed on 25 Nov 1996, which is a continuation-in-part of Ser. No. US 1995-000008, filed on 7 Jun 1995, now patented, Pat. 5,318,931	

DOCUMENT TYPE: 1
PRIMARY INVENTOR: 01 Jerome D.
LEGAL REPRESENTATIVE: 01 James C.; Roof, Carl J.; Graff, Milton B.
NUMBER OF CLAIMS: 1
EXEMPLAR CLAIM: 1

CAS INDEX IS AVAILABLE THIS PATENT.
ABSTRACT: The subject invention relates to compounds having the structure:
 ## wherein n is an integer from 1 to about 3;

with (X and Y are each independently selected from O, S and CH.sub.2,
 and at least one of X and Y being O or S;
 (C is unsubstituted straight or branched chain alkanyl or alkanoxy
 having from 1 to 10 carbon-hydrogen atoms; and
 (C is selected from hydrogen, methyl, cyano, and halo;
 pharmaceutical compositions containing such compounds; and the use of
 such compounds for the diagnosis or treatment of disorders modulated by
 alpha-2 adrenoceptor

CAS INDEX IS AVAILABLE THIS PATENT.
DETD to respiratory challenges such as inhaled citric acid. (See,
 e.g., Callaway, J. Pharm. Med., 1997, 10, 1-10, "Effects of Inhaled .alpha.2-Adrenoceptor
 agonists on Citric Acid-Induced Cough
 and Bronchial Volume in Guinea Pigs", European Journal of
 Pharmacology, Vol. 322, 1997, 1-10. The effectiveness of other
 alpha-2 agonists in the management of neurologic disorders has been
 demonstrated, including attention-deficit hyperactive disorder and
 Tourette's syndrome. See, e.g., Chappell P., M. Riddle, L.
 S. J., K. Lynch, J. J. J., A. Arnsten, J. Leckman & D. Cohen,
 "Clonidine treatment of comorbid attention-deficit hyperactivity
 disorder and Tourette's syndrome: preliminary clinical
 experience", Journal of the American Academy of Child and Adolescent
 Psychiatry, Vol. 36, 1997, pp. 1140-1146), cognitive disorders.

L10 ANSWER OF 45 US A 1
ACCESSION NUMBER: 01 6418 USPATFULL
TITLE: (1-Pazolinylamino) quinolines useful as alpha-2
 adrenoceptor agonists
INVENTOR 01 Thomas Lee, Oxford, OH, United States
 01 Peter J., Cincinnati, OH, United States
 01 Gregory J., Hamilton, OH, United States
 01 Donald T., Pleasant Plain, OH, United States
 01 Russell James, Fairfield, OH, United States
 01 Glen E., West Chester, OH, United States
 01 Sophie E., Maineville, OH, United States

which is effective to reduce or alleviate at least one
 symptoms of Tourette's Syndrome, obsessive-compulsive disorder,
 schizophrenia or other mammal.
 CAS INDEX AVAILABLE TO PATENT.
 DETD has an inhibitory effect of nicotine with respect
 to an enzyme converts glutamate into gammaaminobutyric acid
 (GABA).
 GABA The ratio of glutamic acid to GABA
 (GABA) in the brain plays a crucial role in brain
 excitatory transmitter. GABA is an
 inhibitory transmitter. Furthermore, the concentration of glutamate and
 GABA is higher than they are in the rest of the
 brain. Nicotine to inhibit behavior) which
 has been shown to be effective in treating Tourette's Syndrome, obsessive-compulsive disorder,
 schizophrenia through its ability to
 inhibit the action of GABA so as to make the patient feel better,. . .

L10 ANS OF 45
 ACCESSIO R: 2001 DERWENT INFORMATION LTD
 DOC. NO. 46] WPIDS
 TITLE: GABA-uptake related disorders - used for
 headaches, dementia, alcohol withdrawal
 spasticity, growth disturbances, tardive
 dyskinesia or alcohol abuse.

DERWENT
 PATENT A(S): NO-NORDISK AS
 COUNTRY
 PATENT I. ION:

PAT	KIND	NO	LA	PG
DK	A	19980528		1

APPLICAT TAILS:

PAT	KIND	APPLICATION	DATE
DK	A	DK 1998-727	19980528

PRIORITY INFO: 19980528
 AN 19980528 [46]
 AB DK 27 A U 1 118
 Met the t GABA-uptake related disorders such
 as headac it, alcohol withdrawal symptoms, spasticity,
 gro urban dyskinesia, alcohol abuse, **stuttering**
 , h hes, H chorea, Gilles di le **Tourettes**
 syn incont ictic neuropathy and postherpetic neuralgia.
 Dwg
 AB DK 27 A U 1 118
 Met the t GABA-uptake related disorders such
 as headac ia, alcohol withdrawal symptoms, spasticity,
 gro urban dyskinesia, alcohol abuse, **stuttering**
 , h hes, H chorea, Gilles di le **Tourettes**
 syr incont ictic neuropathy and postherpetic neuralgia.
 Dwg

L10 ANS 0145 COPYRIGHT 2001 BIOSIS DUPLICATE 4
 ACCESSIO 1 BIOSIS
 DOCUMENT 1 511
 TITLE: A patient with glutamate decarboxylase in a patient with
 cerebellar degeneration and Sjogren syndrome.
 AUTHOR(S) 1; Harada, Toshihide; Kamei, Hidekazu;
 Nakamura
 CORPORAT 1: Internal Med., Hiroshima Univ. Sch. Med., 1-2-3
 Kasumi-ku, Hiroshima 734 Japan
 SOURCE: 1: J. Clin. Neurology (Tokyo), (Feb., 1998) Vol. 50, No. 2, pp.
 159-169.

DOCUMENT 1
 LANGUAGE 1
 SUMMARY 1: English
 AB We 1: 52-y old woman with Sjogren syndrome from the age of 46,
 dev cerebe 1: atrophic, autonomic dysfunction and **dysarthria**
 at had n 1: history, and all known causes of cerebellar
 dis re exc 1: of the patient contained autoantibodies
 dir agains 1: against glutamate decarboxylase (GAD) which was an enzyme
 inv in the 1: of GABA. She also had
 aut Abies t 1: specific with Sjogren syndrome (SS-A,
 anti-nuc anti- 1: anti- body changed into negative after high dose
 int and 1: corticosteroid therapy, but symptoms did not
 improve. 1:
 Wes 1: abnormal bands to human neuroblastoma cell
 lin 15, 4% 1: were relatively specific to nervous tissue.

In 1:
 thi cerebe 1: atrophy and atrophy were caused by autoimmune
 pat 1: cerebellar GABAergic system and central nerve
 cel 1:

AB We 1: 52-y old woman with Sjogren syndrome from the age of 46,
 dev cerebe 1: atrophic, autonomic dysfunction and **dysarthria**
 at had n 1: history, and all known causes of cerebellar
 dis re exc 1: of the patient contained autoantibodies
 dir agains 1: against glutamate decarboxylase (GAD) which was an enzyme
 inv in the 1: of GABA. She also had
 aut Abies t 1: specific with Sjogren syndrome (SS-A,
 anti-nuc anti- 1: anti- body changed into negative after high dose.

L10 AL 1: 177 COPYRIGHT 2001 INIST-CNRS. ALL RIGHTS RESERVED.
 ACCESSIO 1: 125 PASCAL
 COPYRIG 1: 1996 INIST-CNRS. All rights

TITLE (1 1: of a Tourette-like syndrome with
 ofloxac 1:
 AUTHOR: 1: J.; REAGAN D. R.; RODRIGUEZ DE BITTNER M.
 1: MARTINEAU P. (trad.)
 CORPORAT 1: 1: Tennessee State Univ., James H Quillen coll.
 1: Dep. internal medicine, Murfreesboro TN
 1: United States
 SOURCE: 1: J. of pharmacotherapy, (1996), 30(2),
 138-141,

DOCUMENT 1: 1: -0280
 1: (case report, clinical case); Translation

BIBLIOGRAPHIE : 1
 COUNTRY : France
 LANGUAGE : French
 SUMMARY :
 AVAILABILITY : 5, 354000052895180050
 AN : 1998
 CP : Co
 ABFR : OF
 OT :
 ol :
 a :
 To :
 a :
 pa :
 l' :
 et :
 l' :
 de :
 co :
 an :
 l' :
 L' :
 co :
 de :
 la :
 GA :
 re :
 as :
 pr :
 ce :
 po :
 l' :
 an :
 as :
 se :
 a :
 ri :
 pr :
 st :
 ABFR :
 d' :
 au :
 si :
 re :
 fut :
 ob :
 co :
 an :
 l' :
 L' :
 co :
 fr :
 po :
 de :
 cas :
 s' :
 l' :
 l' :
 :

IUT-CNRS. All rights reserved.
 relation entre l'emploi de la fluoroquinolone
 et le developpement d'une encephalopathie
 caracteristiques similaires au syndrome de La
 Tourette, relie dans le temps
 avec l'ofloxacin, fut observe chez un
 homme disparut completement apres
 l'arret du traitement. Les caracteristiques les plus remarquables
 de ce syndrome sont la coprolalie ; on a egalement observe de
 des automatismes orofaciaux et des membres,
 l'amelioration de l'annee concernant l'episode pendant la
 clinique avait plusieurs points en commun
 avec le syndrome de La Tourette et possiblement avec de
 l'origine originant du lobe frontal.
 Les examens neuroradiologiques, et du liquide
 cerebrospinal : Les effets neurotoxiques documentes
 de l'ofloxacin, l'insomnie, les convulsions, le delire, et
 les proprietes antagonistes du
 GABA. Il s'agit ici du premier
 rapport au syndrome de La Tourette,
 associe a une quinolone, suggerant une interaction
 avec les neurotransmetteurs dopaminergiques
 centraux. Les effets de medicaments qui, comme l'ofloxacin,
 agissent dans le systeme nerveux central,
 associee aux maladies ou a l'age
 de substances telles la theophylline et les
 stimulants, et possiblement l'augmentation de la
 sensibilite, classent la personne agee dans un groupe
 a risque de toxicite aux quinolones. Les ajustements
 des doses des effets indesirables au niveau du
 systeme central sont primordiaux.
 ABFR. L'ofloxacin chez un homme age et le developpement
 d'un syndrome presentant des caracteristiques similaires
 au syndrome de La Tourette.
 RESUME DU CAS Un syndrome insolite,
 relation d'un traitement avec l'ofloxacin,
 fut observe chez un homme age et le developpement
 d'un syndrome presentant des caracteristiques similaires
 au syndrome de La Tourette, relie dans le temps
 avec l'ofloxacin, fut observe chez un
 homme disparut completement apres
 l'arret du traitement. Les caracteristiques les plus remarquables
 de ce syndrome sont la coprolalie ; on a egalement observe de
 des automatismes orofaciaux et des membres,
 l'amelioration de l'annee concernant l'episode pendant la
 clinique avait plusieurs points en commun
 avec le syndrome de La Tourette et possiblement avec de
 l'origine originant du lobe frontal.
 Les examens neuroradiologiques, et du liquide
 cerebrospinal : Les effets neurotoxiques documentes des
 de l'ofloxacin, l'insomnie, les convulsions, le delire, et la
 les proprietes antagonistes du GABA.
 Il s'agit ici du premier rapport d'un
 cas associe a la Tourette, associe a
 une quinolone, suggerant une interaction possible avec
 les neurotransmetteurs dopaminergiques centraux. CONCLUSIONS

ACCESSIO
DOC. NO.
TITLE:

16 [2] WPIDS
75

ons of sugars and amino acids, opt with other
for treatment of, e.g., Alzheimer's disease,
n, hair loss, cancers, hypertension, etc.

DERWENT
INVENTOR
PATENT A
COUNTRY
PATENT I

T; NAITO, A
NAITO A T; (NAIT-I) NAITO A

PAT

WEEK LA PG

EP

(1995 3)* EN 13

CA

R GB CR IE IT LI LU MC NL PT SE
(199717)#

APPLICAT

PAT

APPLICATION

DATE

EP

EP 1993-308852 19931105

CA

CA 1993-2103399 19931118

PRIORITY

08852 19931105; CA 1993-2103399

AN 199

AB EP

0619

Cor

ery

myo

D(+)

L(+)

L(-)

D(+)

ami

asp

met

tyl

tyl

tyl

tyl

tyl

tyl

tyl

tyl

tyl

tyl

tyl

tyl

tyl

tyl

tyl

tyl

tyl

tyl

tyl

tyl

tyl

tyl

tyl

tyl

tyl

tyl

tyl

one or more pure sugars selected from meso
alactose, D(+)lactose, D(+)xylose, dulcitol,
, D(-)mannitol, sorbitol, D(+)glucose,
se, cellobiose, D(+)maltose, D(+)raffinose,
e, D(-)ribose, adonitol, D(+)arabitol,
L(-)glucose, D(-)lyxose, L(+)lyxose, L(-)lyxose,
amine and D galactosamine; and (b) one or more
glutamine, lysine, arginine, asparagine,
glutamic acid, glycine, histidine, leucine,
alanine, serine, threonine, tryptophan,
ne.

s are capable of passing through the

blood-br

bar

fun

xan

enk

cho

met

B,

thi

uti

bod

use

Hur

cha

sta

anx

dis

lup

sy

f (i) restoring impeded neuro-transmitter
g other materials (e.g. beta-carotene,
cium, somastatin, vasopressin, endorphin,
time, GABA, dynorphin, L-tryptophan,
oxin, niacin, L-arginine, hydroxyproline, NGF,
assium, phosphorus, chlorine, sodium, vitamins A,
phosphate, selenium, linolenic acids, etc.)
rrier, or (iii) otherwise facilitating the
materials in the chemical processes of the
t. together with these other materials) may be
dependence, drunkenness, Alzheimer's disease,
anemia, loss of hair, depression, insomnia,
muscle disorders, stress, headache,
anorexia, panic disorders,
dystrophy, cerebral palsy, Parkinson's
cancers, acne, psoriasis, eczema,
sclerosis, AIDS, chronic fatigue, ageing
also be used in athletic drinks.

AB Dwo
 .
 of
 oth
 son
 GAP
 nia
 pho
 dru
 loss
 of
 dis
 and
 cere
 acc

... neurotransmitter function, (ii) transporting
 ... catecholamine, xanthophyll, lecithin, calcium,
 ... endorphin, enkephalin, acetyl-L-carnitine,
 ... GABA, choline, thiamine, pyridoxine,
 ... oxyproline, NGF, methionine, cystine, potassium,
 ... vitamins A, B, C, K, . . . dependence,
 ... diseases, Huntington's disease, schizophrenia,
 ... chronic pain, osteoporosis, heart muscle
 ... e, **stuttering**, poor memory, bulimia,
 ... ts, anxiety, hyperactivity, muscular dystrophy,
 ... on's disease, hypertension, PMS, shock, cancers,

L10 AND
 ACCESSIO
 DOCUMENT
 TITLE:
 AUTHOR(S)
 CORPORATE
 SOURCE:

... COPYRIGHT 2001 BIOSIS
 ... BIOSIS
 ... 26008
 ... and some related psychopharmacological
 ... Tourette's syndrome: An update.
 ... B. ... Chokka, P. R.; Bornstein, R. A.
 ... Neurochem. Res. Unit, Dep. Psychiatry, Univ. Alberta,
 ... AB T6G 2B7 Canada
 ... of Psychopharmacology, (1995) Vol. 9, No. 3, pp.
 ... 9- 911.

DOCUMENT
 LANGUAGE
 AB Neu
 the
 among
 sex
 whi
 try
 GAP
 op
 mo
 and
 AB Neu
 the
 among
 sex
 whi
 try
 GAP
 op
 mo

... loss of **Tourette's** syndrome (TS) suggest
 ... is disorder may be the result of an imbalance
 ... and/or neuromodulator systems. Neurochemicals
 ... include: catecholamines; acetylcholine;
 ... amines; the amino acids gamma-aminobutyric acid (GABA),
 ... and p-tyrosine; trace amines;
 ... and androgenic hormones. A suitable animal
 ... to advance our understanding of this disorder,
 ... recent developments in this regard.
 ... of **Tourette's** syndrome (TS) suggest
 ... is disorder may be the result of an imbalance
 ... and/or neuromodulator systems. Neurochemicals
 ... include: catecholamines; acetylcholine;
 ... amines; the amino acids gamma-aminobutyric acid (GABA),
 ... and p-tyrosine; trace amines;
 ... and androgenic hormones. A suitable animal

L10 AND
 ACCESSIO
 DOCUMENT
 TITLE:
 treatme.
 AUTHOR:
 CORPORA
 SOURCE:

... E
 ... E
 ... DUPLICATE 6
 ... E
 ... on of **arthria** in multiple sclerosis by
 ... magnetic fields.
 ... Research Laboratories, Danbury, CT
 ... A.
 ... JOURNAL OF NEUROSCIENCE, (1995 Nov) 83 (1-2)

PUB. COMM

LANGUAGE

FILE SE

ENTRY M

ENTRY W

AB

It

sch

so

medic

res

repor

el st

pr

in

patie

dys

with

appli

po

inbl

plea

se of

accl

imed

cha

GABA

AB

It

sch

so

medic

reul

re or

synt

chronic

pr

im

resol

and (

sythe

am no

ce eb

dysar

neuro

th n

Code: ISSN: 0020-7454.

Unit: Kingdom

Article (JOURNAL ARTICLE)

Journal

... or more of patients diagnosed with multiple speech impairment (**dysarthria**) which in ... disabling. Currently there is no effective the **dysarthria** of MS which occurs as a ... cerebellum and its outflow tracts. It was ... extracranial application of brief AC pulsed (EMF) in the picotesla (pT) range intensity ... with MS sustained improvement in motor functions ... otology. This communication concerns two MS ... progressive course who exhibited severe ... disability during the initial treatment ... which solved completely 3-4 weeks later. Since ... been shown to alter: (a) the resting membrane ... neurotransmitter release through an effect ... transmembrane calcium flux; and (b) the secretion of ... in turn influences the synthesis and release of ... amino **butyric** ... it is suggested that the ... **dysarthria** occurred as a result of ... othermitter functions particularly 5-HT and ... amylination.

... at ... or more of patients diagnosed with multiple speech impairment (**dysarthria**) which in ... disabling. Currently there is no effective the **dysarthria** of MS which occurs as a ... cerebellum and its outflow tracts. It was ... motor functions including cerebellar ... communication concerns two MS patients with a ... severe **dysarthria** which ... the initial treatment with pulsed EMFs and which ... weeks later. Since application of ... flux; ... melatonin which in turn influences the ... in (5-HT) and **gamma**- ... in the ... the immediate improvement of the ... of changes in cerebellar ... particularly 5-HT and **GABA** rather

L10 A/SW

ACCESSION

TITLE:

AUTHOR:

LOCATION:

SOURCE:

Ref.

AVAIL. F

LANGUAGE:

DOCUMENT TYPE

Copyright 2001 DERWENT INFORMATION LTD

PT S

... of CNS Stimulants in Gilles de la

...

... V

... om

... (15, No. 5, 408-25, 1992) 2 Tab. 154

ISSN: 0722-5091

... Psychiatry, Middlesex Hospital, London W1N

...

FIELD AVAILABLE

FILE SEQUENCE

AN 1993-

AB The contraindications of stimulants e.g. dextroamphetamine (dexamphetamine), amphetamine, l-amphetamine, or methylphenidate in the treatment of Tourette's syndrome (GTS) is reviewed considering the relationship between attention deficit-hyperactivity disorder (ADHD) and the contraindications to treatment with stimulants. Neurobiochemical systems considered are dopamine (DA), norepinephrine (noradrenaline), 5-HT, ACh, GABA and clonidine. The drugs of choice for GTS are DA antagonists and clonidine. ADHD are stimulants. Stimulants may cause tics, which are weighed up against the benefits of their use. Side-effects of clonidine (DA antagonist) are depression/dysphoria, sedation and hypotension. CL may be the drug of choice for a patient with both ADHD and GTS.

ABEX High doses of stimulants, e.g. pemoline, L-amphetamine and dextroamphetamine, to treat ADHD can provoke or exacerbate tics or GTS in patients without these preexisting symptoms. Tics are

less likely to be provoked by stimulants in patients treated with haloperidol. There is evidence that stimulants and butyrophenones are simply antagonistic to each other but have overlapping effects on tics and side-effects. Levodopa given to patients with tics can result in involuntary vocalization

and apomorphine can create and decrease GTS. Obsessive-convulsive disorder is treated with LSD and psilocin. CL is effective for both tics and GTS but if hyperactivity is a significant problem, treatment with or without butyrophenones/benzamides may be tried in low doses and with relevant monitoring. The neurobiology of GTS is based on the release of dopamine from pre-synaptic nerve terminals. Abnormalities of neurotransmitters such as DA, noradrenaline, 5-HT, ACh, GABA and clonidine (e.g. dynorphin) are noted in GTS. Other drugs used in the treatment of GTS include sulpiride, fluphenazine, naltrexone, clomipramine, methylphenidate, clonazepam, nifedipine, verapamil, fluphenazine, progabide and physostigmine. Other treatments include amitriptyline, desipramine,

imipramine, bupropion, phenhydramine, chlorpromazine, propranolol and carbamazepine (E.C.E.).

AB The contraindications of stimulants e.g. dextroamphetamine (dexamphetamine), amphetamine, l-amphetamine, or methylphenidate in the treatment of Tourette's syndrome (GTS) is reviewed considering the relationship between attention deficit-hyperactivity disorder (ADHD) and the contraindications to treatment with stimulants. Neurobiochemical systems considered are dopamine (DA), norepinephrine (noradrenaline), 5-HT, ACh, GABA and clonidine. The drugs of choice for GTS are DA antagonists and clonidine. ADHD are stimulants. Stimulants may

L10 ANSWER 2

ACCESSION NUMBER

TITLE:

AUTHOR:

LOCATION:

SOURCE:

Ref.

DERWENT INFORMATION LTD

DERWENT INFORMATION LTD

1- Lactam Antibiotics: Predisposing

Authors O; Norrby S R

Sweden

Other. (27, No. 4, 405-25, 1991) 3 Tab. 169

AVAIL. OF DOC.:
Umea,

ISSN: 0305-7453

Pharmacology, University of Umea, S-90185

LANGUAGE:
DOCUMENT TYPE:
FIELD AVAIL.:
FILE SEGMENT:

AN 1991-27570

AB Neurotoxic
benzylpeni
(AM),

oxacillin
ticarcillin
cefonicid
cefotaxime
and imipen
drugs ment

halothane,

propanidid
amphetamin

physostigmine,

isoniazid,
cyclosporin

ABEX Neurotoxic
and epilep

Highest po
meropenem

myasthenia
myoclonus,

delerium,

headache,

recorded f
CX, CD, CU

warfarin,

Drugs know
fentanyl,

enflurane,
theophyllin

phenylprop
metronidaz

and
cyclosporin

sensitivit
are discus

/3H/flunit
cefamandol

ABEX. . . and
myasthenia

myoclonus,
delerium,

headache,
recorded f

to
hexobarbit

In pathoge
GABA, pent
azlocillin

L10 ANSWER 30

Neurotoxic activity, of beta-lactams e.g.
benzylpenicillin (CF), ampicillin (AP), amoxicillin

oxacillin (CO), nafcillin (NC), carbenicillin (CB),
ticarcillin (TP), cefaloridine (CR), cefalotin (CT),
cefonicid (C), cefmetazole (CZ), cefacetrile (CA),
cefotaxime (CD), cefuroxime (CU) and latamoxef (LM),
imipenem and FCE-22101 are reviewed. Other
drugs ment fentanyl, meperidine, ketamine,

propanidid, theophylline, aminophylline,
amphetamin, terbutaline, phenyl-propanolamine,

physostigmine, isoniazid, chlorambucil, misonidazole and

Neurotoxicity by beta-lactam antibiotics occur frequently
and are observed after very high systemic doses.

Drugs with BP, CF and imipenem/cilastin (also
known for more of seizures, aggravation of

symptoms, confusion, nystagmus, encephalopathy,
myoclonus, rapid reaction, **dysarthria**,

delirium, rapid eye movements, tachycardia,
myoclonus and neuromuscular excitability are

reviewed. Factors include concurrent or prior BP,
fentanyl, tobramycin, cefoperazone or amikacin.

Factors lowering seizure thresholds are alcohol,
ketamine, pentazocine, propoxyphene, halothane,

lidocaine, procaine, etidocaine,
amphetamine, ephedrine, terbutaline,

phosphates, physostigmine, isoniazid,
quinolones, chlorambucil, misonidazole

Neurotoxicity in rats and increased
sensitivity to thiobarbital and influence of probenecid

are discussed. Drugs dicloxacillin, phenethicillin,
flunitrazepam, cefmetazole, ceftezole,

ampicillin are mentioned. (E4/AK)

More of seizures, aggravation of
symptoms, confusion, nystagmus, encephalopathy,

myoclonus, rapid reaction, **dysarthria**,

delirium, rapid eye movements, tachycardia,
myoclonus and neuromuscular excitability are

reviewed. Neurotoxicity in rats and increased sensitivity

to thiobarbital and influence of probenecid are discussed.
Drugs, phenethicillin, /3H/flunitrazepam,

cefamandole, ceftizoxime and
azlocillin (AK)

© 2001 DERWENT INFORMATION LTD

ACCESSION NUMBER	1991-12408	AB	Side-effects of various antibacterial drugs on the CNS are reviewed with reference to penicillins, cephalosporins, erythromycin, metronidazole, isoniazid, sulfonamides and tetracyclines.
TITLE	Side-Effects of Various Antibacterial		
AUTHOR	W. H. J. M. S.		
LOCATION	Amst., West		
SOURCE	Suppl. 1, S29-S32, 1991) 87 Ref.		
ISSN	0300-8126		
AVAIL. OF DOC.	Universitaet Goettingen, W-3400 Goettingen, Germany.		
LANGUAGE	English		
DOCUMENT TYPE	Review		
FIELD AVAIL.	1991-12408		
FILE SEGMENT	1991-12408		
AB	Side-effects of various antibacterial drugs on the CNS are reviewed with reference to penicillins, cephalosporins, erythromycin, metronidazole, isoniazid, sulfonamides and tetracyclines.		
ABEX	CNS-effects include fits, headaches, vertigo, visual disturbances, sleeping problems, psychoses, lassitude and confusion similar to those of nalidixic acid, ciprofloxacin, ofloxacin and ciprofloxacin can act through inhibition of the metabolism of theophylline, caffeine and paraxanthine through A-receptors, through interaction with the structural similarities to benzathine-like effects. Quinolone-psychoses can be treated with benzodiazepines can be used in cases of convulsions. Amoxicillin, carbenicillin, ticarcillin, oxacillin, cloxacillin, dicloxacillin, and encephalopathy in adults, and can cause fits and visual disturbances. Metronidazole can cause fits, ataxia, dysarthria and convulsions, ataxia, dysarthria and convulsions can cause vertigo, excitation, cerebral depression, and hyper-reflexia. Sulfanilamide can cause sight disturbances, vertigo, and psychoses. (S67/LJ) (Zentralnervense System und antibakterieller Substanzen.)		
ABEX	... and act through inhibition of the metabolism of theophylline, caffeine and paraxanthine through A-receptors, through interaction with the structural similarities to benzathine-like effects. Quinolone-psychoses can be treated with benzodiazepines can be used in cases of convulsions. Amoxicillin, carbenicillin, ticarcillin, oxacillin, cloxacillin, dicloxacillin, and encephalopathy in adults, and can cause fits and visual disturbances. Metronidazole can cause fits, ataxia, dysarthria and convulsions, ataxia, dysarthria and convulsions can cause vertigo, excitation, cerebral depression, and hyper-reflexia. Sulfanilamide can cause sight disturbances, vertigo, and psychoses. (S67/LJ) (Zentralnervense System und antibakterieller Substanzen.)		
Metronidazole	... can cause fits, ataxia, dysarthria and convulsions, ataxia, dysarthria and convulsions can cause vertigo, excitation, cerebral depression, and hyper-reflexia. Sulfanilamide can cause sight disturbances, vertigo, and psychoses. (S67/LJ) (Zentralnervense System und antibakterieller Substanzen.)		

L10 ANSWER 31
 ACCESSION NUMBER:
 TITLE:
 AUTHOR:
 CORPORATE SOURCE:
 LOCATION:
 SOURCE:
 Tab. 21 R.
 AVAIL. OF DOC.:
 LANGUAGE:
 DOCUMENT TYPE:
 FIELD AVAIL.:
 FILE SEGMENT:
 AN 1989-30106
 AB P.o. vigabatrin administered to patients with drug-resistant complex partial epilepsy. Effects included **stutter**, drowsiness, loss of concentration and irritability and increased vigor.
 VB resulted in a decrease in seizure frequency when administered with drug-resistant complex partial epilepsy. Effects included **stutter**, drowsiness, loss of concentration and irritability and increased vigor.
 returned to pretreatment levels of free and total GABA and HVA. CSF concentrations of homovanillic acid (HVA) significantly after a single dose of VB but subsequent dosing schedules. In contrast, 5-HIAA concentrations increased with the single dose but were not significantly altered by alternate day and daily VB. Drugs included phenytoin (PH), carbamazepine (CB) and p.o. VB.
 ABEX 11 Patients with drug-resistant epilepsy were admitted to the study. 2 Patients were treated with CB alone, 2 with both CB and p.o. VB, 50 mg/kg as add-on therapy. The morning dose. The patients then received 50 mg/kg every other day for a further 2 weeks. The dosage interval was reduced to daily administration. CSF total and free GABA and HVA levels were increased, compared with pretreatment values, 2-fold after a single dose of VB and remained elevated at subsequent dosing. Increases were dose-dependent. VB was administered with every 3rd day dosing and every other day dosing. CSF concentrations of 5-HIAA decreased over pre-treatment levels. A significant increase in values observed after alternate day dosing returned to pre-treatment concentrations. CSF concentrations following single VB dosage. 1 Patient had a decrease in concentration and development of depressive symptoms. 1 Patient reported increased vigor. 1 Reported mild drowsiness. 3/11 patients had a decrease in seizure frequency. 100% while 1 patient increased frequency. 1 Patient had a greater than 50% decrease in seizure frequency in 8/10 cases including

eliminated. In 2. (Y112/SEB)
 AB. adm. with drug-resistant complex partial
 epilepsy. progressively decreased with decreasing
 dosing interval. effects included **stutter**, drowsiness,
 loss of consciousness and irritability and increased vigor.
 VB
 resulted in increases in CSF levels of free and total
GABA and CSF concentrations of homovanillic
 acid (HVA) significantly after a single dose of VB but
 returned to
 ABEX. the dosage interval was reduced to
 daily administration. CSF total and free **GABA**
 and HVA increased, compared with pretreatment
 values, of VB and remained. increased
 following patient had to withdraw because of
 drowsiness and development of a **stutter**.
 Another depressive symptoms and irritability. 2
 Patients 1 Reported mild drowsiness. With VB
 given even

L10 ANSWER 3
 ACCESSION NUMBER 9
 TITLE: 2001 DERWENT INFORMATION LTD
 B V
 acidemia Type I: Effect of Riboflavin and
 AUTHOR: Goodman S I; Batshaw M L
 LOCATION: Durham, Denver, Colorado, United States
 SOURCE: 1, 62-65, 1988) 1 Fig. 1 Tab. 15 Ref.
 ISSN: 0022-3476
 AVAIL. OF DOC.: Institute, 707 N. Broadway, Baltimore, MD
 LANGUAGE:
 DOCUMENT TYPE:
 FIELD AVAIL.:
 FILE SEGMENT:
 AN 1988-104
 AB A 5-yr-old glutaric acidemia associated with
 progressive epilepsy and basal ganglia degeneration and
 who responded (RF) and l-carnitine (CT) with
 evidenced
 of biochemical improvement, is reported. Neither agent
 reduced of acid (GA), but RF increased GABA levels.
 A trial of discontinued because of severe lethargy
 and
 vomiting.
 ABEX The patient until 11 mth of age when he had a 3 day
 episode and low-grade fever that led to seizures
 and
 coma. The viral encephalitis. After this he lost
 some development hypotonic and had choreoathetoid
 movements his skills but at 24 mth, he had a 2nd
 viral-like with severe lethargy and vomiting. CT
 showed brain He continued to lose motor skills
 and
 at 45 mth sturring. He was unable to sit without
 support, hypotonia and choreoathetosis was noted
 on volitional his **dysarthrititis**; IQ was 50-55.
 Glutaric diagnosed on finding peaks of GA (9-24
 ug/ml) and of urine specimens. Plasma GA was 11
 ug/ml. by finding less than 1% of normal

activity in fibroblasts. Magnetic resonance imaging showed bilateral lobe atrophy and fibrosis and hyperlucency of the caudate nucleus. He was put on a protein restriction diet. He received 100 mg/day p.o. RF for 1 wk, 100 mg/kg/day RF+CT continuously. Before treatment, CSF levels of GABA were decreased and HVA and HIAA were normal. RF had no effect on GA but raised GABA to normal. Long term RF+CT treatment was associated with improvement and the rapid progression of the neurodegeneration (E54/RSV) (M.L.B.).

ABEX. hypotonia was diagnosed by GLC/MS. continued on GA but short-chain fatty acids were normal. Long term treatment was

L10 ANSWER
 ACCESSION NUMBER
 TITLE:
 AUTHOR:
 LOCATION:
 SOURCE:
 AVAIL. OF DOCUMENT

LANGUAGE:
 DOCUMENT TYPE:
 FIELD AVAIL.:
 FILE SEGMENT:
 AN 1987-24
 AB Drugs and Parkinson's
 long term use of anticholinergics and other drugs in the treatment of dystonic syndromes, Gilles de la Tourette syndrome and Wilson's disease.
 ABEX Aspects of levodopa therapy are discussed. Levodopa is given with drugs which reduce the peripheral but not the central effects of levodopa.
 levodopa dosage has been optimized. The use of trihexyphenidyl HCl, procyclidine HCl, cycrimin HCl, methixone HCl, orphenadrine HCl is discussed.
 anticholinergics have fewer side effects than levodopa.

bi... are coupled to **GABA** receptors
 an... olytics, muscle relaxants and
 an... T... gent is dependent on pharmacokinetics
 an... ethyl DZ and renal excretion as
 gl... uced sedation are fatigue, somnolence,
 we... hria, ataxia and obnubilation;
 al... ation. Other interactions with tobacco,
 ba... ine, disulfuram, p.o. contraceptives
 and...

L10 ANS... 2001 ELSEVIER SCI. B.V.DUPLICATE 8
 ACCESSIO...
 DOCUMENT...
 TITLE: ... treatment of tardive dyskinesia and other
 AUTHOR: ... n J.E.; Simpson M.L.; et al.
 CORPORATE... psychopharmacology, Palo Alto Veterans
 States... cal Center, Palo Alto, CA, United
 SOURCE: ... ry, (1985) 20/8 (888-893).
 COUNTRY:
 DOCUMENT
 FILE SEGE... ture Index

LANGUAGE:
 AB We... of gamma-vinyl-**GABA** (GVG) in
 nin... dyskinesia, one with Meige syndrome,
 and
 one... tardive dyskinesia patients
 comp... and, as a group, demonstrated
 sig... a scores. Four of these five tardive
 dys... ally evident improvement, with
 app... kinetic symptoms. Other patients had no
 cli... atients had transient exacerbation of
 psy... withdrawal of GVG, and one patient
 exp... al episodes. Our results suggest that
 GAB... treating patients with tardive
 dys...
 AB We... of gamma-vinyl-**GABA** (GVG) in
 nin... dyskinesia, one with Meige syndrome,
 and
 one... tardive dyskinesia patients
 comp... and, as a group, demonstrated
 sig... a scores.. . .

L10 AN... 2001 INIST-CNRS. ALL RIGHTS RESERVED.
 ACCESSIO... PASCAL
 TITLE (I... ABA treatment of tardive dyskinesia and
 AUTHOR: ... disorders
 CORPORATE... HORTON J. E.; SIMPSON M. L.; BERGER P.
 SOURCE: ... M. J.
 2... t., Palo Alto CA, United States
 2... chiatry, (1985), 20(8), 888-893, refs.

BIBLIOGRAPHY

COUNTRY:

LANGUAGE:

AVAILABILITY:

AN 198

ABFR Ana

des

Tou

ABFR Ana

des

Tou

L10 ANS

ACCESSION

DOCUMENT

TITLE:

AUTHOR:

CORPORATION

DK-8000

SOURCE:

COUNTRY:

DOCUMENT

FILE SECT

LANGUAGE:

AB The

to

oper

with

dose

con

trea

invol

with

Gile

with

effe

post

trea

AB The

to

ope

with

of

two

con

L10 ANS

ACCESSION

DOCUMENT

TITLE:

AUTHOR(S)

CORPORATION

SOURCE:

T 2001 ELSEVIER SCI. B.V.DUPLICATE 9

Treatment of hyperkinetic extrapyramidal

E.; Braendgaard H.

ology, Aarhus Municipal Hospital,

Scandinavica, (1985) 72/3 (341-343).

Literature Index

and Neurosurgery

BY

-receptor agonist, progabide,

y movements was evaluated in a preliminary

10 males and 7 females, aged 10-78 years,

patients were included in the study. Daily

0 to 3600 mg (median 2400 mg)

(mean 45 mg/kg), while the duration of

Improvement, with a reduction of

%, occurred in two of four patients

, and in two of three patients

ous, while no consistent beneficial

patients with Huntington's chorea,

on dystonia, tardive dyskinesia, action

to-branchio-respiratory myoclonus.

-receptor agonist, progabide,

movements was evaluated in a preliminary

males and. . . weeks. Improvement,

movements exceeding 25%, occurred in two

la Tourette's syndrome, and in

aroxic intention myoclonus, while no

registered in ten. . .

T 2001 BIOSIS

DUPLICATE 10

TYPE I PRESENTING WITH HYPOGLYCEMIA.

CLASS G J A I

UNIV. LONDON, 30 GUILFORD ST., LONDON

IS, (1984) 7 (3), 122-124.

0141-8955.

FILE SECT
LANGUAGE:

AB A c... enzyme deficiency (type I glutaric
aci... subdural hydromas, and progressive
cho... the diagnosis was made when she
was... at the age of 3.5 yr. Temporary
adr... also noted. Three years after diagnosis
the... hypoglycemia have resolved and treatment
wit... GABA analog, has prevented any
fur... deterioration.

AB A c... enzyme deficiency (type I glutaric
aci... subdural hydromas, and progressive
cho... the diagnosis was made when she
was... at the age of 3.5 yr. Temporary
adr... also noted. Three years after
dia... and hypoglycemia have resolved and
tre... GABA analog, has
pre... deterioration.

L10 ANS... 2001 BIOSIS

ACCESSION...

DOCUMENT...

TITLE: ... AMMA AMINO BUTYRIC-ACID AND ADENOSINE
IN

EPILEPSY... FLUID IN PROGRESSIVE MYO CLONUS

AUTHOR(S)... BIES.

CORPORAL... K; FREDHOLM B B; HARE T A
... LTAVUORENPENGER 10 A, SF-00170 HELSINKI

SOURCE: ... 40 (10), 623-625.
... C003-9942.

FILE SECT
LANGUAGE:

AB Pro... without Lafora's bodies (PME) is a rare
inf... in Finland, where the incidence is
1 c... This fatal disease is characterized by
nor... progressive stimulus-sensitive myoclonus,
ata... grand mal seizures and loss of
cer... concentrations of GABA in the CSF
ave... (4+- SE) in 8 patients with PME,
con... in 10 control patients. The
con... (5 pmol/ml vs. 17 pmol/ml), inosine (560
pmo... inosine (6.2 nmol/ml vs. 6.1 nmol/ml)
we... and in controls.

AB. ... 20,000-30,000 children. This fatal
dis... early development, progressive
str... ia, dysarthria, occasional
gro... cerebellar Purkinje cells. Concentrations
of ... 10 pmol/ml (mean +- SE) in
8 ... 135 +-...

L10 A... 2001 DERWENT INFORMATION LTD

ACCESSION...
TITLE: ... Syndrome.

AUTHOR: ... States
LOCATION: ... 501, 1982)

SOURCE: ...
... 0272-4391

AVAIL. C... try, Texas Tech. University Regional

LANGUAGE:

DOCUMENT:

FIELD A:

FILE SE:

AN 19

AB A

to

cl

sy

co

co

it

ha

ABEX A

with

cl

ab

de

ab

de

In

control

or

ma

AB.

cl

T

s

o

i

a

L10 AN

ACCESSIO

DOCUMENT

TITLE:

AUTHOR(

CORPORAT

BESTA,

SOURCE:

FILE SE

LANGUAG

IT Mi

L10 AN

ACCESSIO

TITLE:

IN

AUTHOR:

LOCATIO

SOURCE:

El Paso, Texas, U.S.A.

C

C

C

C

C

C

C

C

C

C

C

C

C

C

C

C

C

C

C

C

C

C

C

C

C

C

C

C

C

C

C

C

C

C

C

C

C

C

C

C

C

C

C

C

C

C

C

C

C

C

C

C

C

C

C

C

C

C

C

C

C

C

phenobarbital was gradually changed

his Gilles de la Tourette

this case, successful, and should be

the mechanism of CP action in this

could be due to the inhibitory effect

facilitating activity. (congress).

Tourette syndrome had been treated

improvement of the tics and the

phenobarbital was gradually

Improved. Improvement in tics and few

initial CP treatment. Reduction of CP

tics and some grunting sounds.

at bedtime produced remarkable

within normal limits and he has been

during the past 8-mth period.

phenobarbital was gradually

treatment of his Gilles de la

was, in this case,

for clinical trial. The mechanism

not understood, but could be due to the

GNS, and its GABA facilitating

2001 BIOSIS

3

3

CONSIDERATIONS AND A PHARMACOLOGICAL

THE EXTRAPYRAMIDAL SYSTEM.

DOGA; ANGELINI L

INFANTILE, IST. NEUROLOGICO C.

LA EVOL, (1982 (RECD 1983)) 2 (4),

E

E

E

E

E

E

E

E

E

E

E

E

E

E

E

E

E

E

E

E

E

E

E

E

GAMMA AMINO
ACTIVITY STIFFNESS
TOURETTE SYNDROME BALLISM

2001 DERWENT INFORMATION LTD

THE INITIAL PERIOD OF STUTTERING

C

C

C

C

C

C

C

C

C

C

C

C

TR. (77, NO.10, 1555-59, 1977)

isc
 ABEX. Alzheimer's disease.
 ext chorea can be reduced to some
 air (benazine, phenothiazines). Drugs
 isc (sodium valproate and
 uns (L) have been generally
 pil attempts with choline, arecoline or
 inc very briefly. . . . considered
 syndrome, syndromes such as Lesch-Nyhan
 pos (5-hydroxytryptophan), chorea, Gilles
 de tremor (sensitive to
 alc (isoniazid), torsion dystonia and
 Alz

=> log h
 COST IN U

SINCE FILE	TOTAL
ENTRY	SESSION
228.17	232.43

FULL ESTI

DISCOUNT

CA SUBSCR

SINCE FILE	TOTAL
ENTRY	SESSION
-2.35	-2.35